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EPIDEMIC SEPTIC SORE THROAT

I. Historical Review*

BY BENJAMIN WHITE, PH.D.†

EACH succeeding epidemic of septic sore throat establishes this disease more firmly as a definite entity. The whole train of events, including the character of the onset, the nature of the symptoms and sequelae, the type of the infecting organism and the vectors of infection are apparently similar in all the major outbreaks of which detailed studies have been made. Patients stricken with this disease pass suddenly from a state of well-being to a state of prostration with severe aches and pains and fever, all characteristic of an acute intoxication which, in the beginning, is of far greater severity than the local affection in the throat would seem to warrant. As in some other infections with hemolytic streptococci this intoxication may be transient or it may be followed by septic complications. It is a serious disease because of the suffering and deaths it may cause, as well as the economic loss the epidemics entail. Because of the frequent delays in recognizing the disease and the necessity for prompt action in controlling an outbreak, it seems desirable to preface the discussion of the Lee epidemic of 1928 with a review of the more important epidemics which have been previously reported.

The earliest appearance of this disease is unknown. Winslow,[1] in reporting the British literature, cites, among others, some peculiar British outbreaks of sore throat with simultaneous cases of scarlet fever, in which milk or cream might have been the source of infection. One occurred at South Kensington[2] in 1875 where twenty persons were affected. They had all taken milk from a district where there were 119 cases of sore throat. Another happened in Oxford[3] in the spring of 1882, when, among eighty-five persons using a certain milk, fourteen contracted sore throat, six scarlet fever and one diphtheria, while still another was reported from Upton and Macclesfield in 1889.[4]

In 1881 reports appeared of communal attacks of sore throat which, meager as they are in clinical details and lacking any bacteriologic information, were undoubtedly outbreaks of septic sore throat. A note[5] from Aberdeen, Scotland published in 1881 seems worth including here. Three hundred persons were stricken and of 110 families supplied with milk from one source ninety were affected. There were no cases in any family not supplied with milk from this source. The symptoms began suddenly with "a very severe rigor, or series of rigors, which last several or many hours, followed by well-marked febrile symptoms." The throat and tonsils were affected but there were no false membranes. There was swelling of the lymphatic glands in the neck and of the tonsils. The fever generally subsided in two to four days, leaving the patient weak and in a prostrated condition. Relapses occurred and the glandular swelling persisted. There were three deaths, all in "old, frail people." Cutting off the milk supply shortened the epidemic. The note stated, "The disease is not infectious and must be introduced directly into the body."

A similar occurrence took place in Rugby in the same year.[6] Suspicion was directed to milk which came from four dairies, in one of which a cow suffering from garget was found. Three years later[7] an explosive outbreak took place in Dover. Apparently within a very short period of time nearly 300 cases developed, all in persons taking milk from a single dairy. A separate outbreak was traced to the same supply. In addition to the symptoms previously described, there is mention of vesicular eruptions followed either by a raw, red, edematous appearance of the mucous membrane of the throat, or white patches which developed and became chronic. Erysipelas and "purulent formations" were concomitants and in some cases the feet were swollen and painful. There were four and possibly five deaths, two in infants due to toxemia and two in elderly persons suffering from chronic nephritis. The fifth case was one of peritonitis.

Woodhead and Cottrell[8] described the occurrence of sixty cases in ten days in Edinburgh in 1888. Here milk was the apparent source of the infection. Winslow[1] also quotes a report[9] of other cases in Rothesay in 1890.

A small explosive outbreak in Finchley (1895), where cows with teat and udder eruptions were found, was reported by Kenwood.[10] A small group of fifteen persons in Anglesley having "follicular tonsillitis" (probably septic

*From the Massachusetts Department of Public Health, Antitoxin and Vaccine Laboratory.

†For record and address of author see "This Week's Issue," page 839.

sore throat) due to milk infected with streptococci has been recorded by Grey-Edwards and Severn.[11] In the same year fifty cases of sore throat occurring in Surbiton on one milk route where a milker having tonsillitis was discovered are described by Coleman.[12] Between that year and 1900 there apparently are no reported outbreaks, but in the latter year Warry[13] studied over 155 cases in North Hackney. Of these, 138 were supplied by one milkman, but the source of the infection was not found. Newsholme[14] reported eighteen cases in Brighton (1901) where the situation was complicated by the presence of scarlet fever. In 1902 there were more than seventy-five sick with this disease in Lincoln. From Mair's [15,16] description the symptoms seem somewhat atypical, but it was probably septic sore throat and again was caused by milk. A Bedford outbreak of forty-two cases, all milk-borne, was reported by Nash.[17]*

In 1904 a more serious epidemic struck Finchley. The disease appeared to be wholly characteristic and between January 16 and January 30 affected 550 persons. Kenwood[18] traced the infection to a dairy and made the important observation that on this farm *there were two cows showing no signs of mastitis, but which gave a curdled milk containing streptococci* (Italics ours).

The Glasgow cases of 1904 reported by Chalmers[19] may be mentioned, although the clinical and epidemiological descriptions are scanty. There, also, milk was supposed to carry the infection, and it may be significant that here a new cow suffering from an eruption of the teat was discovered. There was the unusual observation that this mammary eruption spread among the other cows in the dairy affecting thirty per cent. of the herd.

Robertson[20] describes a prolonged appearance of cases of diphtheria and sore throat breaking out in Leith from June to October, 1904. Although no relationship was established between the cases and the milk, such a source seems probable. At least, five cows were found in a dairy with ulcers on their teats.

The first really significant report is that of Pierce,[21] describing an epidemic occurring in Woking (Surrey) early in October, 1903. In all there were 250 cases with eight deaths that were undoubtedly caused by the infection. Because this is the first fairly complete account of the characteristic symptoms it may well be included here:

> **The sore throats varied in character and extent. Many resembled follicular tonsillitis, while in others there were confluent pieces of membrane, and still others were like quinsy. There was sudden and marked constitutional disturbance, with high temperature, headache, pain in the back and limbs and general malaise. There was a strong tendency to the involvement of the submaxillary and post cervical glands which, if they did not break down, remained persistently enlarged. In some cases the pharynx and larynx were involved, while one nasopharyngeal case developed pyemia resulting in death. This patient's wife developed symptoms of puerperal fever. Sometimes the tissues about the tongue become swollen. There was arthralgia in some instances. A few cases of erysipelas occurred, some very serious, extension taking place from the nose and mouth and in some by the way of the Eustachian canal to the outer ear. One case of mastoiditis terminated fatally on the tenth day. Seven other deaths were registered as "pneumonia and cardiac failure," "toxemia, coma and septic hypostatic pneumonia," "ptomaine poisoning, arthritis and pleuropneumonia," and one "acute ulcerative colitis and peritonitis."**

The milk from one farm was involved and in the dairy herd one cow was found that had had mastitis in one quarter for two to three years. *Another cow showed one affected quarter that had not been detected by the milker, and which was only recognized when the milk was drawn into a shallow container. Only then was it noticed that the milk from this quarter was pinkish and contained curds.* (Italics ours) From this milk two strains of streptococcus were isolated, but the description of their characters permits no definite indentification. Pierce, however, mentions a fact which, in the light of our subsequent knowledge, may be of genuine importance. He found that the farmer connected with the suspected milk supply had a severe attack of sore throat about the middle of September and that on the twenty-ninth he complained of joint pains. The query arises was the farmer's infection the source of the cow's infection or could he have directly contaminated the milk supply? The presence of streptococci in the unnoticed infected quarter may well have been the cause of the outbreak, but whether there was a connection between the farmer's sore throat and the cow's mastitis must remain a conjecture.

Robb (1905)[22] describes the Paisly experience in which over one hundred cases occurred. The milk was suspected and it was shown that it had been obtained from cows suffering from cowpox. In the same year Savage[23] reported a series of typical cases from Colchester. Here also the milk was incriminated, a cow with mastitis being found and streptococci isolated from the milk. Again it was noted that the farmer's was the first case to develop, followed in four days by the appearance of mild sore throat in his wife and children, but the interval between the farmer's infection and the twelve cases in Colchester seems to be too short to account for a man-to-cow-to-milk-to-human train of infection.

These various British epidemics are more fully described by Swithinbank and Newman[24] and Savage,[25] but have been presented here in abstract form in order to show the long continuance of the disease in England and Scotland and to bring out various facts which seem to be of importance in the light of subsequent outbreaks.

*In the Lancet, II, p. 1291, 1902, Savill compares the Bedford outbreak with one occurring in certain metropolitan infirmaries in 1891 (see Trans. Med. Soc., London, XV, p. 103, 1892) and Lunn states that a disease of this kind has recurred every autumn for six years, with one year's remission. He believes them to be due to milk. (S. & N., p. 359.)

Although no search has been made of the European literature, the paper by Sömme[26] may be mentioned here to show that this disease has occurred in epidemic form in countries other than Great Britain and America. This author describes an outbreak of a considerable number of cases in Christiania in 1908. Among 548 cases notified, Sömme found that three times as many adults as children were affected. There were some deaths. The majority of the patients took milk from one dairy on which a cow was found with a diseased udder giving milk containing a streptococcus similar to that isolated from the patients' throats.

In Denmark, Larsen[27] studied an outbreak of 840 cases developing in Kolding, a town of 20,000 inhabitants, between December first and twentieth, 1926. At first, as sometimes happens, the disease was supposed to be influenza, and such a long interval elapsed between the appearance of the first cases and official notification to the State health authorities that a complete and satisfactory survey was impossible. There were eleven cases of erysipelas, forty-seven of otitis, and thirty of rheumatic fever. The eleven deaths were mainly due to edema of the larynx, and erysipelas. Cultures showed that a hemolytic streptococcus was the causative organism. Inquiries brought out the fact that all cases took milk from one diary. Owing to the lateness of the study it was impossible to trace the infection to any individuals or cows. Larsen estimates that in all nearly 2400 persons were affected.

The first serious epidemic to appear in this country, or at least the first to be carefully studied, occurred in Boston, Brookline and Cambridge in May, 1911.* Winslow[28] discusses the symptoms as follows: "The onset of the attack was rapid and often accompanied by severe headache and acute grippy pains. The temperature was high, often 103° to 105°. The throat was somewhat variable in appearance. At first there was a general diffuse redness extending over the tonsils and pharynx and adjacent regions, much like scarlet fever throat. Later small isolated patches of white usually appeared, resembling the plugs of ordinary follicular tonsillitis. Still later in many cases there was an extensive membrane-like exudate simulating diphtheria. Frequently one tonsil was swollen first, followed by the other, and there was almost always more or less invasion of the cervical lymph nodes.

"This first stage of the disease lasted for three to five days and was followed by recovery or by secondary complications. In either case the patient usually experienced great prostration. The commonest complications were abscesses in the peritonsillar region and in the cervical glands. The infecting organism, whatever it was, did not stay long in the throat, and affections of the nose and ear were relatively rare. It was the deeper tissues that were affected and abscesses produced were exceedingly painful and difficult to handle. Operations often failed to locate any pus, and after a time the abscesses discharged themselves, but the neck remained in a painful condition for a long period afterward."

*An outbreak occurred in April, 1908, among Harvard students all eating at Memorial Hall. While examination of the milk showed streptococci, the data are too incomplete to be of value. Report, Special Milk Board, Mass. Dept. of Health, 1916, p. 255.

In still more serious cases complications of a more general septic nature followed. The occurrence of these acute complications was closely dependent upon the general condition of the patient. With children and young people the whole course of the disease was light; but with the old or those of low resistance, almost any weak organ might be attacked. Rheumatism was perhaps the commonest complication. Erysipelas was another. Nephritis occurred in a number of instances. Pleurisy and pneumonia in many cases followed the initial attack. The heart was frequently affected. One patient had in succession, following her sore throat, erysipelas, pneumonia, and pericarditis. In the most dangerous cases of all, including many of the fatalities, there was a general systemic infection, leading to septicemia or peritonitis.

The total number of cases collected by Winslow was 1,043, although the total number of persons affected was probably about 2000. There were forty-eight deaths that might be attributed to this infection. It was found that over eighty-four per cent. of the cases appeared on the list of a single dairy, and eight per cent. more were believed by physicians to have used this milk. In addition to these cases there were 392 in Hudson, Marlboro and Southboro where milk from the same dairy was apparently the main factor. Winslow's investigation fails to reveal any infection among the cows but he found that cases of tonsillitis had occurred at "the proper time" in a family on this farm and in families of employes of the dairy. There is no mention in the report of any bacteriologic studies of the patients, milk or cows. In this outbreak the milk came from one of the best dairies in the state, where the most modern methods of sanitary milk production were practised, save one, and that was pasteurization.[29, 30] Shortly after the outbreak just discussed, a severe epidemic took place in Chicago (December, 1911 to March, 1912). Detailed reports have been made by Davis and Rosenow,[31] Miller and Capps,[32] Capps and Miller,[33] Heinemann,[34] and Capps [35, 36] on the clinical and epidemiological aspects of the outbreak; and Davis,[37] and Capps and Davis[38] on the bacteriologic features. It was estimated that over ten thousand persons were stricken. The disease was unusually severe and was followed by a variety of complications. In addition to the symptoms previously described by other authors, the following observations were reported by the Chicago physicians: The prostration and constitutional symptoms were out of all proportion to the local involvement. The pulse was relatively slow, due probably to a toxic irritation of the vagus nerve. Leucocytes were only moder-

ately increased. A greyish diphtheria-like membrane was sometimes present but ulceration of the mucous membranes of the throat was rare. Relapses were frequent and were attended by such complications as suppurating lymph nodes, conjunctivitis, empyema, arthritis, erysipelas, purpura, acute fibrinous pleurisy, bronchopneumonia, pulmonary abscess, endocarditis, otitis media, brain abscess, meningitis, nephritis, septicemia and puerperal sepsis.

The great majority of the persons affected were supplied by the same dairy, and in the herds of this dairy (including 1848 cows) there were eighty-five cases of mastitis in December, January and February. On eleven of the farms supplying this dairy there were twenty-nine cases of sore throat, and on nine of these farms there was also mastitis among the cows. On one of these farms a sixteen year old girl was very ill with sore throat on December 18th. On another farm supplying the same dairy there was a cow suffering with mastitis of five weeks' duration. Her milk showed streptococci, and streptococci were found in a culture taken at the same time from the throat of a girl on the same farm who, four weeks before, had been afflicted with sore throat and arthritis, but who denied having drank any milk. There were many features disclosed in the investigation which abundantly showed the possibility of the transmission of streptococcal infection from a human source not only to the milk but to the cows.

The milk from this dairy was usually pasteurized by the "flash" method but there were pronounced failures of the process on December 17, 19, 28, 30 and on January 7 and 11. The explosive outbreak began on December 21, reached its climax on Christmas Day and rapidly subsided. A second explosive outbreak was on January 1 and 2, while a smaller eruption of cases occurred from January 10 to 17. Allowing from twenty-four to thirty-six hours for delivery of the milk and thirty hours to four days for the incubation period of sore throat, the bursts of cases correspond to the breaks in the pasteurizing process.

Davis'[37] study of the hemolytic streptococcus isolated from typical cases gives us the first detailed description of the organism which we know as Streptococcus epidemicus and which has been found to be associated with practically all subsequent epidemics.

While the situation in Chicago was clearing, Baltimore was afflicted with a similar visitation (February, 1912). Hamburger[39] says that at first the disease was indefinitely described as "grip" but that later it became apparent that they were dealing with "a new and terrible disease." Children were chiefly affected but adults were not spared. Vomiting was common at the onset and the other symptoms were similar to those already described. The usual complications ensued while the majority of deaths were caused by peritonitis. A more complete report of the Baltimore outbreak was made by Frost, Stokes and Hachtel[40]. They state that certainly more than 1000 and probably 2000 or more persons were stricken, while records of twenty-eight deaths were obtained. As in Chicago, a hemolytic encapsulated streptococcus was regularly isolated from patients and was identified as Streptococcus epidemicus[41 42].

In this same period a milk-borne infection caused an estimated total of about 1000 cases in Concord, New Hampshire. Mann[43] studied the circumstances surrounding 472 cases and found that the majority of these persons took cream and milk from one source which he believed was infected by a milk handler. Here also, as in Chicago, the milk was pasteurized by the "flash" method, and the pasteurizer had not been functioning properly for some time before the epidemic began. The ultimate source of the infection was not definitely determined, and although there were numerous cases of sore throat among the dairy employes, it is unlikely that such a long continued infection could come from accidental contamination of the milk from human beings. It is far more likely that infected cows supplied the streptococci.

There was also an outbreak, with cases mainly among children in Philadelphia in February and March, 1912. This infection, too, was milk-borne and was traced to a milker[44].

Ice cream, made from apparently infected milk caused sixty cases in Middlebury, Vermont, but the probable source of infection was not reported[45].

In late April, 1913, the disease appeared in epidemic proportions in the towns of Cortland and Homer in New York State. With a combined population of 14,700 there were 669 reported cases of septic sore throat, with fourteen deaths. The fatalities were due to peritonitis (5 cases), erysipelas (4 cases), pneumonia (2 cases), heart failure (2 cases), and edema of the larynx (1 case). The outbreak was explosive, but was preceeded two months previously by a few scattered cases of sore throat in both towns. This fact may be of importance. North, White and Avery[46], in reporting this epidemic, state that seventy-two per cent. of the cases were found among the patrons of a single dairy and that this dairy delivered less than seven per cent. of the total milk supply. In this herd two cows were found to be suffering from garget, while another had a slight infection of one quarter. The milk from these animals and from an apparently healthy cow all showed the presence of hemolytic streptococci. It is difficult to reconcile these authors' bacteriologic findings with those of previous reports because there was considerable variation in the characters of the different strains isolated, and only one of the strains produced typical hemolysis when added to washed human or sheep erythrocytes. There is no suggestion that human beings contributed streptococci to the milk either directly or indi-

rectly. The milk alleged to have caused the illness was raw milk.

In the same and the following year outbreaks occurred in the towns of Norton, Canton, Wakefield, Stoneham, Winthrop and Westfield, Massachusetts[47 48 49 50]. In Wakefield and Stoneham it was estimated that nearly one thousand persons were ill and their sickness was attributed to a single supply of raw milk. Morse[49] states that on the farm from which the milk came a milker had had tonsillitis with a peritonsillar abscess twenty and again twelve days before the outbreak. No more cases appeared after pasteurization was instituted. Smith and Brown[50] in discussing their bacteriologic findings in the first three outbreaks say that they found hemolytic streptococci similar to those reported in the Boston, Chicago and Baltimore epidemics in the Canton and Wakefield-Stoneham cases but a somewhat different strain in the peritoneum and throats of several Norton patients. In this instance the strains isolated from the throats of three patients and from the peritoneum of another were not of the Epidemicus type and were not similar to the strains isolated from the cows or the suspected milk.* Some of Smith and Brown's conclusions, because of their importance, may well be quoted here:

"I. The streptococci causing epidemics of tonsillitis are not necessarily the same in different epidemics either in the same or different localities.

"II. The success likely to attend the tracing of such epidemics to their source will depend upon a minute, detailed study of individual strains of streptococci and the discovery of certain minor distinguishing characteristics as guides.

"III. The streptococci which have been the agents of recent outbreaks are all alike in that the colonies produce, immediately around them, a clear hemolyzed area on blood agar plates (horse blood). They differ from the common throat coccus, our *a* type, in which the colony has a partly discolored and hemolyzed mantle between it and an outer narrow clearer zone.

"IV. There is at present no satisfactory evidence that bovine streptococci associated with mastitis or garget are the agents of tonsillitis in man. Whenever cases of garget are suspected as sources of infection in man, both human and bovine types should be looked for."

Bray[51] reported forty cases of tonsillitis in Ray Brook, New York, (1914) caused by raw milk coming from a farm on which one of the milkers was suffering from sore throat. In the same month thirty cases, all on one milk route supplying raw milk, occurred in Elmhurst, Illinois. Rosenow and Moon[52] isolated from the milk hemolytic streptococci which closely resembled the throat culture strains in their main characteristics.

In the spring of 1914 a series of nearly 300 cases reported as septic sore throat and due undoubtedly to infected raw milk occurred in Dutchess County[53], New York. A member of a milker's family, suffering from a sore throat may have been the original source of infection. This outbreak is mentioned because the question was raised whether the infection was that of septic sore throat or atypical scarlet fever. We have plenty of evidence that scarlet fever in epidemic proportions may be caused by an infected milk supply, and now we know that we may have scarlet fever with no rash. Those who study future epidemics of sore throat should bear in mind the similarities and dissimilarities of infections caused by hemolytic streptococci.

Of 232 cases studied in an outbreak at Rockville Center, New York (June, 1914) Overton, Krumwiede and Jacques[54] found that eighty-nine per cent. were on a single milk route supplying raw milk. Krumwiede and Valentine[55] in their bacteriologic study of the outbreak isolated streptococci similar culturally, and identical in their agglutinations from cases of septic sore throat and *from the udder of one cow showing no evidence of mastitis, except the peculiar character of the milk from one quarter.* (Italics ours.) They concluded that the infection was primarily of human origin and was due to a streptococcus of the Epidemicus type. The importance of their study lies in the fact that a cow may be giving milk containing hemolytic streptococci of human origin and capable of causing a serious epidemic with so little evidence of teat infection that the usual milker would fail to detect its presence and might include the infected milk in the regular supply.

Capps and Davis[56] studied an epidemic which took place in Jacksonville, Illinois. They investigated 348 cases which were traced to ice cream and milk from a dairy where cows affected with a streptococcus mastitis were discovered.

Other outbreaks were reported in 1914. At Easthampton, New York, forty-six cases were traced to milk[57], while other cases were reported from Carbondale, Illinois, due also to infected milk[58]. In the winter and spring of 1915 the disease appeared again in Massachusetts. Smillie[59] described a localized outbreak in a boarding school due to milk that had been improperly pasteurized. Hemolytic streptococci of the Beta type were isolated from the cases and from the udders of the cows. In the next two months 310 cases were reported in Milton and Dorchester[60 59]. Over ninety per cent. of the cases in the two towns took milk from one distributor. On the farm supplying this milk, cases of sore throat and one of scarlet fever were found, and a cow with garget and another showing pus in the milk were discovered. There again a hemolytic streptococcus of the Beta type was isolated from patients and cows. In May, 1916, Watertown, Massachusetts, had forty-six cases traced to raw milk from a farm where a carrier of hemolytic streptococci and a cow with the same organisms in the udder were found[60]. In May of that year eighty-six cases developed in West

* "In only one of the epidemics (Canton, Mass.) reported by Smith and Brown can it be said that the same streptococcus found in the patients was also isolated from the suspected milk. The other epidemics were not proven bacteriologically to be of milk-borne origin." Brown, J. H., Frost, W. D., and Shaw, M.: Hemolytic Streptococci of the Beta type in certified milk. Jour. Inf. Dis., XXXVIII, 5, p. 381, May, 1926.

Winfield, New York, all in users of raw milk or cream from one dairy in which a similar ambulant case occurred[61]. Bridgeport, Connecticut, in June had a sizeable epidemic (400 cases)[62]. Here again ninety per cent. of the patients took raw milk from one dairy. A severe case in a bottle handler at the beginning of the outbreak was supposed to be the source of the infection. In January to March, 1917, about fifty cases developed in Needham and Newton, Massachusetts. Kelley[60] reports that the raw milk used by these patients came from a dairy on which there were two milkers with hemolytic streptococci in their throats and a cow with a similar organism in one quarter. In February and March of this same year an explosive outbreak of 325 cases (in a total population of 941) took place in Galesville, Wisconsin. Henika and Thompson[63] say that the cases were largely among users of milk or ice cream from one dairy, and on this dairy one dairyman had a severe case of sore throat January 28. Several carriers were found on this farm and identical streptococci were isolated from the carriers and the milk. Six cows were found with streptococci in their milk, three of them with mastitis, but the other three showed no signs of garget as the term is usually understood. Rosenow and Hess[64] describe such unusual symptoms in these Galesville cases as small superficial ulcers of the uvula and pillars, with edema of the eyes and eyelids, myositis and synovitis. Ninety-five per cent. of the sick took the contaminated milk which was found to contain a highly virulent streptococcus.

Next came the Brookline-Dedham epidemic which was so completely studied by Brown and Orcutt[65]. The milk came from a model dairy where unusual precautions were taken to insure the quality of the milk but where pasteurization was not employed. The disease was moderate in extent and severity and was characterized by clinical symptoms somewhat different from those usual in septic sore throat. The organism isolated from the patients and from a single quarter of one cow was Streptococcus epidemicus. While a number of milkers on this farm were found infected, it was impossible to trace the infection of the cow's udder to any one of the milkers, though the authors feel that such an infection was probable, since the streptococcus isolated from the cow was in every respect like streptococci isolated from patients and milkers, and different from those found in normal cows or cows with garget. Then came the outbreak in Somerville and Medford, Massachusetts[60], in which forty-two cases were reported but with an estimated total of 150. Three employes of one milk handler were sick and one had hemolytic streptococci in his throat. Kelley[60] reported 150 cases in Gloucester, Massachusetts (April and May, 1917) all taking milk from one route. On one of five farms supplying this dealer a housewife was found who had hemolytic streptococci in her throat. In Massachusetts again (July-August, 1917) three neighboring towns, Wellesley, Natick and Dover had 119 cases due apparently to milk from a dairy in which there were two milk handlers with hemolytic streptococci in their throats[60]. St. Albans, Vermont, in the same year had 1200 cases believed to be due to milk contaminated by infected milk handlers[66], and a similar source is mentioned by Kelley and Webber[67] as causing forty-three cases in Winchester, Massachusetts (June-July, 1920).

In February, 1919, there was an outbreak of hemolytic streptococcal infections of an unusual and hitherto unreported nature occurring in the Chelsea (Mass.) Naval Hospital. Keegan[68] describes simultaneous cases of what was undoubtedly septic sore throat and surgical wound infections due to a hemolytic streptococcus. The cases of sore throat appeared first in the nose and throat surgical wards and then spread to the general surgical wards. Here the throat affection would develop one or more days after the operation and from two to four days later the wound would show infection. During the first twelve hours of the attack the symptoms were those of influenza, but soon a moderate leucocytosis could be noted. The rise in temperature lasted for two days, with frequent relapses and an occasional septic complication such as erysipelas, otitis or sinusitis. The organisms isolated from the throats of the sick and from the infected wounds were apparently identical and were undoubtedly Streptococcus epidemicus. Altogether more than one hundred cases were observed but no connection between them and the milk supply could be found. The milk contained no streptococci, and its general use by patients was not followed by sore throat attacks. Keegan believes that it was a direct contact infection.

Apparently there were no other epidemics between 1917 and June, 1920, since no reports for this interval were found; nor between 1920 and 1922. In March of this latter year typical cases of septic sore throat appeared in Portland, Oregon. Within a period of little more than a week 487 cases developed, all referable to the milk of one dairy. There were twenty-two deaths, and we find in addition to the usual causes of death mentioned in other epidemics such diagnoses as acute encephalitis, myocarditis, appendicitis and septic cholecystitis. Benson and Sears[69] who investigated the situation, succeeded in obtaining similar strains of hemolytic streptococci, all of human type, from the inflamed udder of a cow of the herd, from one milker's throat and from the throats of numerous septic sore throat patients and contacts. They say that it is probable that the milker in question infected the udder of the cow.

In the next year (May, 1923) sixty-eight cases

were reported from Arlington, Massachusetts. Here once more milk was implicated and a case of sore throat was found in a member of the producer's family[67]. In July thirteen cases, reported by Laidlow[70] occurred in New York State, and in October seven cases in Harwich, Massachusetts, are included in the Kelley and Webber report. In 1924 one outbreak of eighty-nine investigated cases was reported from Danbury, Connecticut[71], fifty-two of which were on a single milk route distributing raw milk from a farm on which a case in a milker and also a gargety cow were found. At Logan, Ohio, (October, 1925) the health officers investigating an outbreak recorded the histories of 295 cases. About sixty per cent. of the cases took raw milk from a dairy which supplied only ten per cent. of all the milk distributed in that town. A streptococcus was isolated from the throats of patients and convalescents but was not identified nor was its source determined.[72]

Frost and Carr[73] describe an outbreak of sixty-three cases with no deaths in Madison, Wisconsin in April, 1926. No cultures apparently were taken from any of the patients, but typical cultures of Streptococcus epidemicus were obtained from two persons on the dairy farm supplying the bulk of the milk consumed by those stricken. One of these individuals was a milker who, although he declared he had never had sore throat, showed an inflamed throat on examination. Moreover, it was this man who regularly cared for and stripped the particular cows which came under suspicion. Two cows were found with Streptococcus epidemicus in their milk. In one the udder infection was acute and 36,000,000 streptococci were found in each cubic centimeter of milk. When the cows were kept in a pasture, the streptococci disappeared but three months later reappeared and persisted for an additional two months. These authors state that cows once infected with Streptococcus epidemicus may remain as sources of infection for long periods of time and probably should never be returned to the milking line.

Another Connecticut outbreak occurred in Guilford in August, 1926, totalling 220 cases. A single source of raw milk was given as the vector of infection.[74] These cases were followed in the next month by eighty similar cases in towns near Guilford and New Haven, traceable to certified milk (raw) from a farm on which there was a case of sore throat.[74]

The most recent epidemic is that which occurred in Lee, Massachusetts in July, 1928. There, in a village of 4000 inhabitants there were 777 individuals who complained of being ill. In addition there were eighty-seven non-resident cases, and still another sixteen that are not included in the first figure. Lombard[75] estimates that in all between 925 and 975 persons were affected. The outbreak was sudden and, because of the symptoms, for a week it was thought that the disease was influenza. There were forty-eight deaths. The infection came from raw milk, and was traced to a single cow, which had a mastitis in one quarter, but which was found to be excreting hemolytic streptococci of the Epidemicus type also from another and apparently healthy quarter. Here once more the very important fact was disclosed that milk heavily laden with disease producing streptococci may come from a teat which shows no grossly visible signs of infection. For a detailed epidemiological report of this epidemic the reader is referred to Lombard,[75] for the bacteriological study to Robinson and Beckler,[76] while the clinical symptoms are described on page 805 of this issue by Holmes.[77] A resume of the more important features may be found in the article by Bigelow and White[78] on page 807 of this same issue.

SUMMARY

A study of the available literature brings out the following facts:

1. Epidemic septic sore throat is a definite disease, caused by a hemolytic streptococcus usually of the Epidemicus type, but rarely of a non-identical, but closely related type. It is almost exclusively milk-borne but infection by contact though unusual is not impossible.

2. The disease may be frequently diagnosed as tonsillitis and more often as influenza.

3. The symptoms are characteristic of a hemolytic streptococcal infection. At first there is a severe and acute intoxication, sometimes disappearing abruptly with no sequelae. Often there is a remission followed by all the septic complications which virulent hemolytic streptococci are capable of causing.

4. Different outbreaks vary in extent and severity, depending upon the number of consumers of the infected milk, the age of the consumers, possibly upon the virulence of the streptococcus, and upon the promptness of the measures taken to remove or destroy the infection in the milk supply.

5. In the majority of outbreaks the infective milk or cream was raw, and in the few instances where supposedly pasteurized milk was distributed it has always been found that this process was not properly carried out.

6. The best evidence shows that the sources of the infection is one or more quarters of cows' udders harboring or propagating hemolytic streptococci of a human—not bovine—type, and what is more important, this infection may exist and persist without producing any systemic symptoms in the cow or even any of the more conspicuous signs of mastitis. Streptococcus laden milk may, therefore, be drawn from such an infected quarter and be included in the supply without attracting the attention of the milker.

7. While some authors claim that human carriers or persons having or recovering from sore

throat may directly contaminate the milk supply, it seems more and more probable that they pass their infection to the teat of the cow and that the cow's udder may become the reservoir of these streptococci.

8. There is little direct evidence bearing on the manner in which the infection survives in the inter-epidemic periods. We may assume either the existence of human or bovine carriers or that the pyogenic hemolytic streptococci of the ordinary sore throat may on transference to the udder of the cow take on new characters and an increased virulence for man.

These collected experiences are valuable in that the information gained shows the way in which the danger of possible future outbreaks may be lessened. They are:

1. The sudden appearance in any community or region of an unusual number of cases of sore throat accompanied with severe prostration should lead immediately to a search for a possible relation between the cases and the milk supply, and if an undue proportion of cases occur on one route or from one source, prompt steps should be taken to pasteurize the milk or to correct any faults of the pasteurizing process if employed.

2. Immediate search should be made for cows suffering from any inflammation of the udder or giving milk which shows any abnormal characteristics such as the presence of curds, pus, hemoglobin or blood, and any such cows should be immediately isolated and the use of their milk discontinued.

3. Dairy cows producing milk for human consumption should be regularly and frequently examined for possible infection and particularly for any affection of the udder and teats. This can be accomplished by manual and visual examination in severe cases, but in many instances only by examination of the milk by collecting it in shallow containers, or better by passing it through a fine-meshed strainer. Such careful inspection combined with frequent bacteriologic examination of the milk from each cow would go far to prevent these outbreaks.

4. One is forced to the conclusion that raw milk, even when produced under what are supposed to be ideal conditions, may carry virulent streptococci to man and that proper pasteurization still remains our greatest safeguard against these diseases.

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EPIDEMIC SEPTIC SORE THROAT

II. A Clinical Study of the 1928 Epidemic in Massachusetts

BY MAY SALONA HOLMES, M.D.†

EPIDEMICS of septic sore throat are not met with in the experience of the average physician. The early recognition of the existence of such an epidemic is so important to the public health that a clinical description of the disease as it was manifested in the recent epidemic in Massachusetts in 1928 is worth putting on record.

The epidemic started in the latter part of June and the peak was reached early in July. The cases personally observed were those admitted to the Emergency Hospital which was opened on July 8th and some seen in consultation. Although most of the persons attacked had become sick prior to the opening of the hospital, enough early cases were seen to illustrate the early stages of the disease.

During the month of May there had been an unusual amount of moderately severe "influenza" of the bronchial type. Whether this had any relation to the later epidemic of septic sore throat is problematical. Another point in the histories of the children affected with septic sore throat was noted by Dr. Sylvester, who saw some 70 to 80 of these cases and says: "I was impressed by the frequency with which parents told me of a preceding illness, five or six weeks before, in which the child was acutely ill for several days and had lumps in his neck. In addition to this, parents told me of at least a dozen cases of severe sore throat with tremendous prostration, preceding the epidemic by several weeks." Among the adults there were some who gave a history of having had the earlier "influenzal" affection, but there were also some who had not had it.

The clinical picture of the disease in children was unlike that in adults. The children were usually only mildly ill and seemed to have either follicular tonsillitis or scarlet fever. Had these cases not occurred during the epidemic they would not have excited comment; but they are linked with the cases in adults by the finding of Streptococcus epidemicus in throat cultures taken from some of them.

In adults the disease was characterized by sudden onset of the usual symptoms of an acute infection headache, malaise, fever, chills, boneaches, and especially prostration. This latter symptom was at all stages of the disease unduly great in comparison with the other symptoms and signs. Sore throat was present at the onset of about 90 per cent. of the cases.* There was one peculiarity of the early throat symptoms which has not been mentioned by previous observers. This was a severe boring pain beginning inside the larynx. Sometimes this was the first symptom observed and was usually accompanied by a spasm of the throat, making swallowing difficult, coming on suddenly and without prov-

†For record and address of author see "This Week's Issue," page 839.

*Where figures or percentages are given, they refer to the seventy-five hospital cases except when otherwise noted.

ocation, and sometimes waking the patient from sleep.

The buccal and pharyngeal cavities presented a picture of intense oedema, unaccompanied by membranes or ulcerations. In the moderately sick individuals, this oedema was confined to the palate, but in the more severe cases the pharynx and larynx were also involved. In examining the throat, one saw a glistening, glairy palate, usually intensely red; but the redness was not as sharply circumscribed as in scarlet fever. The uvula was round like a marble. In only one case was the tongue involved. The tonsils were seldom much enlarged unless there was a pre-existing chronic enlargement; but as the anterior and posterior pillars of the palate became engorged the tonsils were pushed forward, giving a false impression not only of tonsillar enlargement but also frequently of peritonsillar abscess. In only one of the hospital cases was there anything resembling a membrane. In this patient there was a film like white of egg extending over one pillar, which, although it persisted many days, was not accompanied by evident ulceration and could be brushed away.

If one could see past the palate into the pharynx, there was the same glistening, glairy appearance. The epiglottis was also large and round. In two throats, I had an opportunity to make digital examination when attempting relief by intubation. I found all landmarks obliterated; the tissues were firm from the intense engorgement but very smooth and slippery; the epiglottis was like a marble, and back of it was a large round ring with a dimple indicating the position of the laryngeal opening. Intubation tubes slipped in easily but as quickly slid out. There was nothing to grip them. Oedema of the glottis was present in eight of the hospital cases; and by mechanical obstruction was the immediate cause of death in several cases outside the hospital.

Accompanying the oedema of the throat there was also marked swelling of the tissues of the neck. In some instances this gave a bull-neck appearance. Enlargement of the cervical glands was not universal nor was it proportional to the throat involvement. Thirty of our hospital cases showed cervical adenitis, usually unilateral, but in only three did the process go on to suppuration and require incision. Resolution of the adenitis was very slow.

As in most acute infections, there was considerable variation in the height of fever. In most of the hospital patients it did not exceed 103°, although one (a fatal case) had a temperature of 105.2°.

Relapses were not uncommon. Many patients felt enough better after a few days to get up and around and then became worse again. Several patients were admitted to the hospital because of recurrence of symptoms.

An erysipelatous type of cellulitis occurred in twenty of the cases, in sixteen on the face and in four on the extremities. Except that the lesions were not so sharply outlined nor quite so intense in color, they closely resembled erysipelas. In one patient with cellulitis of the leg, suppuration eventually occurred. Three of these patients died.

The only other skin eruption noted was morbiliform and occurred in only one patient, lasting for 48 hours after admission on the fifth day of his illness.

Arthritis was noted in five patients, three of whom had had antitoxin. In no instance did the arthritis go on to suppuration.

Glycosuria, which is not mentioned in earlier accounts of this disease, was found in 42 (56%) of the cases. One of these was a known diabetic and two others had had diabetic symptoms, but had not had their urine examined previously. These three died. In the others, the glycosuria was transient, being controlled by diet in all but five, and by diet and insulin in these. We do not know whether the glycosuria was renal or pancreatic but incline to the latter view because of the slight evidence of kidney damage by this infection.

White blood counts showed a leucocytosis, varying from 12,000 to 50,000, with from 80 to 92 per cent. polymorphonuclear neutrophilic leucocytes.

Complications were surprisingly few in this epidemic, if we accept cellulitis as part of the usual course of the disease. Peritonitis, frequent in the 1911 epidemic, was noted in one hospital case and, so far as can be learned, in only three others. The hospital patient was a man of 55 years with a slight throat lesion, in whom peritonitis developed a few hours before death. Of the others, one was a man of 69 years, one a boy of 17 and one a girl aged nine. She was sent to another hospital and was operated on for appendicitis but was found to have general peritonitis. All four of these persons died.

Quinsy was not observed. One patient had a retro-pharyngeal abscess.

Lesions of the lung were rare and occurred only as terminal conditions. The same is true of the heart, there being a few cases of pericarditis or endocartitis. Nephritis was not observed except in those who had pre-existing kidney lesions.

Otitis media, evidenced by earache and reddening of the drum, was found in 13 cases, but in only four was drainage required. Mastoiditis was not observed.

Presumably illness lasted longer in the hospital cases than in those who were treated at home, so that figures for duration of the disease based on hospital cases may give a false idea of the severity of the infection. However, the following figures are not without interest. For non-fatal cases admitted on the first to third day of disease, the average duration of disease up to the

time of discharge from the hospital was ten days. For similar cases admitted later than the third day, this period was increased to three weeks. In the fatal cases, deaths occurred on the seventh, eighth (two), ninth, tenth, and twelfth (two), twenty-first and twenty-sixth days.

Although the epidemiological and statistical aspects of the epidemic are taken up in detail by Lombard,[1] it is worth noting that the deaths occurred in the aged patients. Of the 75 hospital cases, only 29 were 50 years old or older; but all the deaths were among these 29. Of the nine fatal cases, five were 75 years or more old. Three others were diabetic and over fifty, leaving only one, a man aged 50, who might have been expected to have enough reserve power to carry him through an acute illness. Pre-existing infirmities were obviously a determining factor in the fatal outcome of most of these cases.

Treatment was supportive and symptomatic. Most important was nursing care. Elimination was augmented by diaphoresis, diuresis and catharsis. As sedatives the coal tar derivatives or any of the newer preparations, supported by generous doses of brandy, were of value. Opiates were harmful. Caffeine proved to be the best stimulant. Adrenalin seemed to increase dyspnoea and was soon abandoned. Locally, hot applications were more effective and more comfortable than cold.

Serum treatment was tried, using scarlet fever and erysipelas antitoxins, but it was not of demonstrable value.

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EPIDEMIC SEPTIC SORE THROAT

III. Review of the 1928 Epidemic in Massachusetts*

BY GEORGE H. BIGELOW, M.D., AND BENJAMIN WHITE, PH.D.†

SINCE 1911 the Commonwealth of Massachusetts has suffered from fifteen outbreaks of septic sore throat. Whether affecting small or large groups, whether occurring in summer or winter, this serious disease has shown a consistency of character that should make its future recognition and arrest easy and prompt, even if the people fail to take the lesson to heart and neglect the one precaution—pasteurization—necessary for its prevention. In order that the circumstances of this most recent outbreak may be made common knowledge to the medical and public health professions in New England, and in the hope that these circumstances may lead to the proper safeguarding of our milk supplies, this review of the Lee epidemic is presented. The main facts are these: In a town of 4000 inhabitants, within a period of about two weeks nearly 950 persons were stricken with septic sore throat and 48 persons died as a result of it. The cause was Streptococcus epidemicus carried by raw milk from a cow suffering from an udder infection with this organism.

The outbreak began about June 29, 1928, reached its peak on July 1 and rapidly subsided after July 8. The disease in its attack on the individual was as acute and severe as in its attack on the community. Persons in their usual good health felt suddenly ill and were quickly prostrated. With adults the first symptoms were those of a toxemia and were quite reasonably mistaken for those of influenza. In children they were less severe and resembled those of the first stages of tonsillitis or scarlet fever. This sudden burst of cases with early symptoms, so alike, and so like the well-remembered signs of influenza, led the local physicians to believe that they had to deal with epidemic grip, and the early outside opinion they sought confirmed this diagnosis. The possibility of a milk-borne infection was, therefore, not entertained at first. The persistent and increasing edema of the throat, the appearance of a few ear infections and inquiries into the possible causes led to the suspicion that the disease was septic sore throat and that milk was responsible. The clinical histories, as described by Holmes,[1] tally with those described in other outbreaks. There might be a short uncomplicated toxemia followed only by more or less debility, or there might be a remission, then exacerbation accompanied by the usual virulent streptococcal sequelae. The deaths were mostly among the aged or infirm, although, when this streptococcus succeeded in passing the barrier set up by the cervical lymph nodes, it killed in a few instances by setting up a general sepsis, finally localizing in the peritoneal cavity.

A hasty investigation by the authors on July 7 established the diagnosis and revealed the fact that the great majority of persons stricken had taken milk from one distributor. The discovery of earlier and some continuing cases of sore throat among the personnel of this distributing plant led to a tentative assumption that the source of the infection might be found there. While the milk of this dealer was later shown to be the undoubted cause of the outbreak, the source of the infection was evidently a cow and not the affected milk handlers. The knowledge that raw milk was the usual vector in such outbreaks led to the order by the local board of health that all milk henceforth sold in

*From the Massachusetts Department of Public Health.
†For record and address of author see "This Week's Issue," page 839.

the town must be pasteurized or boiled and also that a house to house notification be given that any milk on hand should be boiled before use.

Lombard,[2] from the analysis of his epidemiological study, found that sore throat prevailed in the community in May and early June, which may or may not be of significance; that over ninety per cent. of the cases occurred within an interval of two weeks; that of 777 cases studied 565 were regular patrons of Distributor "A", while 158 more were occasional patrons, although this dealer supplied only thirty-five per cent. of the milk sold in Lee; that, although apparently fewer children than adults were stricken, and slightly fewer males than females, computations made on the exposed group only showed no significant differences between the sexes and age groups; that the incubation period averaged two days; and that contact was probably responsible for less than five per cent. of the cases. In addition to the cases in the town of Lee, information was obtained regarding eighty-seven non-residents who, during shorter or longer stays in that town received the infection there but developed the disease elsewhere. From the information gleaned from these histories it would seem that a single drink of the infected milk was sufficient to cause this illness.

On July 10 veterinarians inspected the various dairies, samples of milk of grouped cows were taken for bacteriological examination and further cultures from the throats and ears of patients were taken and studied. It was soon found that a hemolytic streptococcus was common to the cases and to the milk from one group of cows on Dairy "D" supplying Distributor "A." At this dairy a cow was found with active mastitis of the left front quarter. This condition had existed prior to the epidemic but the dairy owner stated that no milk from this quarter had been included in the supply. However, bacteriologic examination showed that besides this quarter, the apparently healthy left hind quarter was also giving milk containing hemolytic streptococci. This animal was isolated, and through the kindness of the owner was acquired and kept under observation until September 14. Robinson and Beckler[3] describe their finding during this period as follows: "The first samples were obtained July 14 and this organism was obtained from both quarters of the udder. Samples were then taken at intervals and S. epidemicus obtained from both left but neither right quarters up to and including September 11. On September 12, 13 and 14, plates from milk from the left hind quarter failed to show S. epidemicus, indicating that bacteriologically the infectious process in this quarter had cleared up.

"The appearance of the milk obtained from the different quarters showed changes corresponding roughly to the pathological changes present. On July 14, samples from the right front and hind and the left hind quarters appeared grossly normal, although that from the last named actually contained S. epidemicus at that time. This is of great importance for it demonstrates that a teat may be pouring out S. epidemicus without any gross changes appearing in the milk or in the cow. The milk from the left front quarter could be called milk only because it was a fluid obtained from a mammary gland. It was yellow, purulent and thick, showing some tendency to separate on standing. All the samples obtained from this quarter, including the final one on September 14, were of this character and were so thick that bacterial counts could not be done satisfactorily. By July 27 milk from the left hind quarter resembled that from the corresponding front quarter but was not then or later so grossly purulent. Plate counts were made on it at intervals up to September 14. Apparently the two right quarters were never involved in the infectious process, for all samples of milk from them were normal in appearance and were negative for the presence of S. epidemicus."

The source of the cow's infection could not be determined. One of the employes at this dairy had suffered from sore throat late in May followed by pneumonia early in June, and shortly afterwards two of his children had sore throat. A culture from one of these children taken in July gave a hemolytic streptococcus which did not resemble the streptococcus from the infected cow. While an attempt to infect a normal quarter of a healthy cow with this organism was successful to the extent that the infection persisted for two months and led to a mastitis, yet the organism remained true to type and developed no new characters that would ally it with the organism associated with the epidemic. A continuation of this study is being carried out by one of the authors.

In their bacteriological investigation, Robinson and Beckler studied cultures taken from the throats of patients ill with the disease, from two ears where otitis was a complication, from the blood in a fatal case and from repeated samples of milk from the two infected quarters of the isolated cow. Cultures were also taken from all persons handling milk to be sold in Lee. For comparison three strains of Streptococcus epidemicus (one from a patient in the 1911 Boston epidemic, one from the 1912 Chicago epidemic and one from a cow from the 1917 Boston outbreak), three cultures of bovine streptococci, all supplied by Dr. J. Howard Brown, and other strains of S. hemolyticus were put through the usual cultural, biochemical and other tests. These authors found that the majority of the strains isolated from the throats of patients, those from the ears, that from the blood, and those from two left quarters of the infected cow corresponded in all their characters to those of Streptococcus epidemicus. This organism they

describe as follows: "a hemolytic streptococcus, growing on the surface of fresh blood agar as moist, sometimes irregular colonies which dry out rapidly if incubation is too prolonged, possessing virulence for mice, fermenting dextrose, lactose, saccharose and salicin, but not mannite or raffinose, bile-insoluble, producing acid to a pH not lower than 5.0, and possessing a capsule when examined by India-ink methods." These bacteriological findings, in a convincing manner, link the cow with the cases, and although the source and manner of the cow's infection remain a mystery, it is entirely reasonable to conclude that it was a hemolytic streptococcus of the Epidemicus type, of human origin, propagated in a cow's udder and conveyed through raw milk, which caused the sickness of this group of nearly one thousand persons, and that it was the same variety of streptococcus that was responsible for the previous Boston, Chicago, Baltimore and many other similar disasters.

As a result of this experience two pasteurizing plants have been installed in Lee and on February 11 of this year an ordinance was passed requiring pasteurization of all milk sold in public places within the town.

This epidemic at Lee in its clinical, epidemiological and bacteriological feature was a typical outbreak of septic sore throat. The suffering and deaths it caused, the immediate cost and the economic loss not only to Lee but to the Berkshire region and to the whole State of Massachusetts are of course incalculable. But the tragedy will be still greater if we fail to appreciate its portent and to do everything possible to prevent a repetition of such a calamity.

When we realize that this disease has afflicted this one state fifteen times in eighteen years, that its symptoms and manner of spread have been repeatedly described, and that we have a sure and relatively inexpensive means for preventing this infection, it is shocking and humiliating to find that not yet can many of our physicians and health officers recognize septic sore throat when the first cases appear, that they are slow to apply common epidemiological knowledge and that they and the public fail to give disinterested and aggressive support to established measures for making milk a safe article of food.

From 1911 to 1928 inclusive, the period comprising this State's experience with septic sore throat, there have been reported only 369 cases of *water-borne* typhoid fever, while there have been 1,468 cases of typhoid fever, 74 of diphtheria, 1,147 of scarlet fever and 3,986 cases of septic sore throat reported as having been caused by *milk!*

We guard our public water supplies with scrupulous care, and we no longer tolerate polluted water, but any attempt to apply our modern knowledge of sanitation to milk is often met with a discouraging and amazing lack of sympathy or encouragement on the part of the consumer and sometimes by a militant opposition from the very group—the producers and distributors—to whose interest it is to make a safe product. We have ordinances and statutory regulations governing the amount of solids and butter fat that milk shall contain, we prohibit the addition of water and we specify this and that grade for milk of higher or lower quality but, in spite of many a grievous experience and in spite of the possession of a full knowledge and ample and proven means, we fail utterly to prevent the spread of communicable diseases by milk.

The contemplation of this one state's record of milk-borne disease since 1910 should cause one to demand that conditions which lead to such suffering, loss of life, and economic waste should no longer be permitted to continue.

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FOR THE DOCTOR'S WAITING ROOM

We reproduce herewith the text of an attractive card with an ingenious device to permit the same being hung on the wall or placed on a table or mantlepiece, which the New York Diphtheria Prevention Commission will shortly mail to all practicing physicians in the city. There is a space for the doctor's signature. Commissioner Wynne in a covering letter will ask all physicians to help in the fight against diphtheria by displaying this card prominently in their waiting rooms.

TO PARENTS

I am asked by the Commissioner of Health to call your attention to the importance of having all children between nine months and ten years protected against diphtheria with toxin-antitoxin. This is simple, safe and lasting. The doctors are co-operating with the Health Department to wipe out this dangerous disease of children. If you have children who have not yet been protected, please have this attended to.

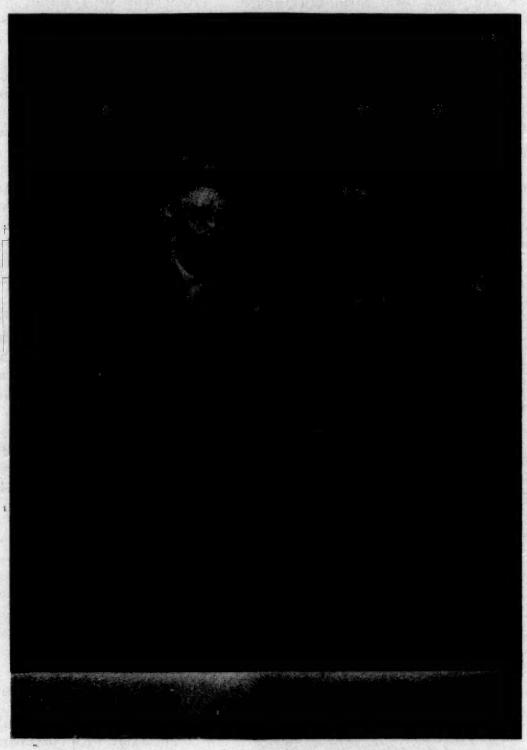
M. D.

—*Bulletin N. Y. Dept. of Health.*

EXERCISES IN CELEBRATION OF THE BICENTENARY OF THE BIRTH OF JOHN HUNTER*

DR. CUSHING—

IN this year of 1928 three notable centenaries have fallen. Three hundred years ago was published Harvey's imperishable tract on The Motion of the Heart and Blood, the cornerstone of modern physiology. The same year (1628) saw the birth of Malpighi, founder of microscopic embryology and histology who among other things discovered the capillary link in the blood circuit. Harvey, as has been said, made the existence of this link a logical necessity; Malpighi, an histological certainty. John Hunter, born just a hundred years after Malpighi, in his turn discovered the collateral circulation and incidentally got his name forever attached not only to a fascial canal in the thigh but to a type of operation for aneurysm. This, however, was by no means his chief reason for fame; what is known as "the Hunterian method" was a far more important contribution for it revolutionized surgery.



JOHN HUNTER.
(Painted by Sir Joshua Reynolds, and Engraved by William Sharp.)

Harvey's concise treatise on the circulation will stand for all time as a model of presentation—the product of many years of thought and experimentation. It completely overshadows his other published work, that on generation, dragged out of him reluctantly, but which in many respects was a no less notable contribution to knowledge. The dictum, *ex ovo omnia*, we accept as one of the basic principles of biology;

*A brief account of the exercises before the Harvard Medical Society, held at the Peter Bent Brigham Hospital, December 11, 1928, when the principal speakers were Dr. Leroy M. S. Miner, Dr. William Fearce Coues, Dr. Arlie V. Bock, Dr. Frederic T. Lewis, and Professor William M. Wheeler, with Dr. Harvey Cushing presiding. A display of Hunter's editions, English and American, with portraits and Hunteriana of various kinds was arranged by Dr. Henry Viets.

Note: Reference to each of the authors appears on page 839.

but Harvey was not a microscopist and it remained for Malpighi to found the science of embryology. Hunter, too, in his turn was deeply interested in and spent years of study on the development of the chick concerning which he doubtless knew much more than is indicated by his fragmentary notes that have been preserved. So these three famous men touch common ground in their speculations on generation as well as in their contributions to our understanding of the movement of the blood.

Very different men they were—the patient, dignified, scholarly Harvey—the sensitive, loveable, fair-minded Malpighi—the self-educated, uncouth, aggressive and quarrelsome Hunter, who undoubtedly of the three had the greatest native genius. It is strange that in the case of each of them some of their work, and one always fears their best work, was lost to posterity —by the mischance of a civil war in Harvey's case, by a conflagration in that of Malpighi, and by Everard Home's unforgivable treatment of the mass of notes and manuscripts that came into his hands after Hunter's death.

With the irreparable loss of the Hunter manuscripts, one may contrast the preservation of the note books in which were set down the speculations and observations of a still greater and more original mind—Leonardo's. He too was interested in the movement of the blood and in generation but he published nothing, and his great fame would have rested almost wholly on the tradition of contemporary reputation were it not for the fortunate preservation of his commonplace books. We shall never know exactly what were John Hunter's speculations on what we now call evolution and on geology and on embryology and on the many other things to which he applied himself.

Though the Valley of the Clyde has given to the world many distinguished men, Hunter eclipses them all. He was a rebellious youth who hated school, loathed books, and saw no good in the kind of learning handed out even at Oxford in the couple of months he wasted there long after his boyhood was over.

"When I was a boy [he said in later years], I wanted to know all about the clouds and the grasses, and why the leaves changed colour in the autumn; I watched the ants, bees, birds, tadpoles, and caddisworms; I pestered people with questions about what nobody knew or cared anything. They wanted to make an old woman of me, or that I should stuff Latin and Greek at the University; but these schemes I cracked like so many vermin as they came before me."

Certainly an unpromising youth, this, whose insatiable curiosity about Nature was a fault, not a virtue. He was the youngest of ten children—ten years younger than his distinguished brother William, a man of entirely different mould, polished and scholarly with all the social graces, already making his mark in the English metropolis as had many Scots before him. At twenty, John in his turn took his way to London to become his brother's apprentice in the private school of anatomy being conducted there. In these surroundings with the structure of the human body to study, the lad's curiosity over things living and dead was aroused to the highest pitch; and in the effort to get answers to the questions his inquisitive mind suggested, he began to show those astonishing powers of application that ere long saw him forging ahead in an independent career which has wholly eclipsed that of his brother. Their lives nevertheless are so inextricably intertwined one can scarcely speak or think of one apart from the other. They unhappily fell out before the end —a sad chapter in the story of the Hunter brothers we need not dwell upon in this celebration.

The years passed and Hunter began to put his seething ideas about natural phenomena on paper. He was made a member of the Royal Society (1767), even before his brother, and articles from his pen, badly expressed many of them, soon began to appear in the Transactions of that distinguished body. His bibliography covers an amazingly varied number of subjects. The first paper was on *The Descent of the Testis,* an anatomical study which had of course an important bearing on the etiology and treatment of hernia; the second was on *The Absorption by Veins;* the third was *An Account of an Amphibious Bipes;* the fourth was a far more pretentious work, a *Treatise on the Natural History of the Human Teeth*—it was the first of two large quarto volumes to be published on the subject. I know of no one who has ever read these tomes, nor what Hunter contributed to the subject of the teeth, nor how he got led into it, but I have asked the Dean of the Dental School if he will not tell us.

DR. MINER—The first serious work published by John Hunter was his "Natural History of the Human Teeth". This appeared in May, 1771, and was not only of scientific importance, but also seems to have been of material aid to Hunter, for it is said that the proceeds of this publication, which came out only two months before his marriage, were appropriated to defray the expenses attendant upon that event.

In Ottley's Life of Hunter that precedes Palmer's edition of Hunter's works, we find the statement that this treatise was

"the first work on the subject which, in addition to an accurate anatomical description, furnishes comprehensive views of the physiology and pathology of these organs".

Not only is the structure of the various parts of a tooth described, but the embryology of the teeth is given consideration. The physiology of occlusion, general comparisons in function in young and old people, bone growth, a subject now given a great deal of attention by the orthodontists, all receive attention.

Hunter's attitude toward this work on teeth is reflected in a statement made by Thomas Bell, one of Hunter's assistants. Bell says,

"That this state of a branch of practice as susceptible of connection with physiological and pathological science, and as improveable by such a connection as any other, should have early attracted the attention of a man preeminently qualified for detecting and supplying such deficiences, and whose labors, unparalleled as they are for their scientific importance, are not less valuable for the immense influence they have since exerted upon the practice both of medicine and surgery, might have been anticipated, from the peculiar character of his mind, which was too truly great to think any subject unworthy of his anxious attention which involved the improvement of the art of healing or the extension of our knowledge of Nature's operations."

It is interesting in this work on teeth, "to trace the peculiarities of Hunter's genius and to watch the workings of his mind in his search for truth, which he pursued with an ingenuousness and candor which have never been surpassed and rarely, if ever, equalled."

Many of Hunter's observations, made as early as 1755, indicate his keenness of observation, his breadth of learning and his zeal for truth. In discussing the structure of the bony part of the teeth, now called dentine, he says,

"We cannot by injection prove that the bony part of a tooth is vascular, but from some circumstances it would appear that it is so. . . From experiments it would appear that the teeth are to be considered as extraneous bodies with respect to circulation through their substance, but they have most certainly a living principle by which means they make a part of the body and are capable of uniting with any other part of a living body."

While Hunter could not prove by experiment that the teeth have a living principle, his surmise that this must be the case has been substantiated since. It is a fact that in these last few years the matter of circulation in the dentine and enamel has been studied by men in this country and in England and it is now well established that both the dentine and enamel have a modified form of circulation.

In 1778 Hunter published the second part of his work on the teeth. This part treats of the diseases of the teeth and adjacent parts. The introduction is quite striking:

"The importance of the teeth is such that they deserve our utmost attention, as well with respect to the preservation of them when in a healthy state, as to the methods of curing them when diseased. They require this attention not only for the preservation of themselves as instruments useful to the body, but also on account of other parts with which they are connected, for diseases in the teeth are apt to produce diseases in the neighboring parts, frequently of very serious consequences, as will evidently appear in the following treatise.

"One might at first imagine that diseases of the teeth must be very simple and like those which take place elsewhere in the bony parts of our body, but experience shows to the contrary. These diseases, considered abstractively, are indeed very simple, but by the relations which the teeth bear to the body in general, and to the parts with which they are immediately connected, they become extremely complicated."

These statements might well be a preamble to a present-day paper on dental focal infection or other subject dealing with the biological relationships between the teeth and the body as a whole.

"The Decay of the Teeth Arising from Rottenness—Diseases of the Alveolar Processes and the Consequences of Them—Scurvy in the Gums, Vulgarly So-Called—Nervous Pains in the Jaws—Extraneous Matter upon the Teeth—Irregularities between the Teeth and Jaws—Supernumerary Teeth—Transplanting and Replanting Teeth" are some of the subjects dealt with in the second part of the treatise. Bell gives Hunter credit for having originated the practice of transplanting teeth, and while this practice has been entirely discontinued, it is of importance as it is related to the early history of the transplantation of tissues.

Hunter quite obviously not only had the conception that diseases of the teeth cause disturbances in other parts of the body, a matter strongly emphasized today, but there is a suggestion that he also had an idea that the reverse of this might be true, namely that physical deficiencies produced trouble with the teeth. He says,

"Whichever of the connected parts be originally diseased, the teeth are quickly the greatest sufferers. None of these parts can be distempered without communicating to the teeth such morbid effects as tend to the destruction of them."

This aspect of dental pathology is not fully appreciated even today.

Hunter's observations that diseases in the teeth are apt to produce diseases in other parts of the body were essentially disregarded for more than a century. Not until what we might call a biological Renaissance in dentistry, beginning approximately in 1900, were the teeth rediscovered as having important relationships to other parts of the body. Dentistry today, in our better university dental schools, is being developed along biological lines with mechanics the servant rather than the master of biological principles. It is clear that Hunter had this idea strongly developed and it seems a pity that it has taken us more than a century to appreciate the truth of what he had to say.

DR. CUSHING—Dr. Miner has alluded to the legend that the "Treatise on the Teeth" which was the only one of his works Hunter sold to a publisher, was thus disposed of to defray the expenses of his wedding which occurred shortly afterward. The evening before the event he wrote the following letter:

Jermyen Street, Saturday Evening.

Dear Brother,—To-morrow morning at eight o'clock and at St. James's Church, I enter into the Holy State of Matrimony. As that is a ceremony which you are not particularly fond of, I will not make a point of having your company there. I propose going out of Town for a few days; when I come to Town I shall call upon you. Married or not married, ever yours,

JOHN HUNTER.

As William did not sign the register with the bride's parents, we may surmise that he was not present. Sunday morning at eight o'clock was perhaps too much for him. As for John, he

probably could not bring himself to give up a week-day from his work, even to get married. The event had been postponed, so it is stated, owing to the disease which Hunter had inadvertently communicated to himself in May, 1767, the complete cure of which, he says, occupied three years.

To return to Hunter's bibliography as given by Palmer, we find that after the publication of the 1771 volume on the teeth, there came a series of papers mostly published in the Transactions of the Royal Society on a most varied number of topics: On the Digestion of the Stomach after death; Anatomical Observations on the Torpedo; Certain Receptacles for air in Birds; Observations on the Gillaroo Trout; Experiments on Animals and Vegetables with respect to the power of producing heat; Proposals for the recovery of people apparently drowned; An account of the Free Martin; The Organ of Hearing in Fishes; Observations on the inflammation of the internal coats of Veins; and so on. Certain of these articles with many additional ones on subjects no less varied were published from Hunter's house in 1786 under the title of *Observations on certain parts of the Animal Œconomy;* and in the same year was issued also from his private printing establishment the equally large quarto entitled *A Treatise on the Venereal Disease,* of which Dr. W. P. Coues will give an account.

Dr. Coues—John Hunter on the Venereal Disease, 15 minutes! It can't be done! But as the late Dr. John Homans used to say, when confronted with a particularly large and difficult ovarian cyst. "Let us make an effort."

London, the time, 1788. A medical student comes out of Nichols' book stall, under his arm a new book. He walks rapidly down the narrow dark street and turns into another, still more narrow, to his lodgings. He runs up the rickety stairs, two flights, to his little back room, lights two tallow candles on his rough desk, and starts reading. Soon he becomes absorbed. Thoughts of "Polly Pattens, adored by half the youth of Guy's," soon vanish, and also those about that smart slashed and spangled costume that he is about to bargain for with Sammy Moss, in which he may seek the favor of Polly's glances at the masque. He reads on; the room gets cold and the candles sputter; he snuffs them and returns to his book, intent on John Hunter of St. George's "Treatise on the Venereal," just out.

Boston, 1928. An out-patient surgeon at one of our large hospitals is demonstrating a lesion of the lower lip to a group of students. Says he: "This lesion is indurated at the base,—the typical Hunterian induration; it is discrete, the submaxillary glands are enlarged and very hard. This has all the characteristics of a primary lesion of the lip, as described by Hunter, but we are not sure. Let the patient be sent to the North Medical department for a dark field examination. A little later the blood Wassermann or Hinton test will be positive, and we shall have the clinical, microscopic, and serologic tests all in accord."

How simple! In the same way, in the genito-urinary department, the diagnosis of specific urethritis is confirmed as a routine by microscopic examination in hundreds of cases every day. Again, how simple! To the younger physician and student of today, all this accurate knowledge concerning venereal disease seems fixed,—it seems as if it had been so for ever. In reality, the time of this accurate knowledge, judged in terms of medical "light years" is but a few days, or a few hours. It is difficult for any of us to realize that the picture-puzzle was not correctly put together in its entirety until the discovery of the spirochete by Schaudin and Hoffman in 1905.

In the time of John Hunter and before, up to the era of Philippe Ricord, the whole subject was in a state of controversy, fierce medical fights were being waged in England and on the Continent about the question of the etiology of gonorrhoea and syphilis, and the different ulcerations, and whether they were due to the same cause or not.

Notwithstanding the result of the unfortunate and tragic experiment which Hunter performed upon himself, the treatise on venereal disease was one of his greatest works, for in it are truths concerning many of the controversies which raged. They clarified and made apparent much that was in a fog and cloud of medical superstition and inaccuracy. To get a clear idea of this, the various English editions, and particularly the French edition of the Treatise, with the exhaustive notes of Richelot and Ricord must be read and compared. Babbington's introduction should not be missed. He says: "Of all the works of John Hunter there is none on which he bestowed more labor or which he was more solicitous to perfect than his 'Treatise on the Venereal Disease'. 'I am resolved,' said Hunter to a friend, 'that it shall not be a mere booksellers' job, every subsequent edition rendering the former useless. The truth of the doctrines I have proved so long as to reduce them to conviction, and in order to render the language intelligible I meet a committee of three gentlemen to whose corrections every page is submitted.'" The names of the three physicians were Gilbert Blane, George Fordyce, and David Pitcairne. The third edition of the book was published after Hunter's death by his brother-in-law, Sir Everard Home, and Babbington says that in it are some passages which were certainly never written by John Hunter. Certainly today the part of the book concerned with diseases of the bladder and prostate, showing so much knowledge on conditions then so little understood, seems the most remarkable. In this connection Babbington's remarks in the introduction are of interest.

He says: "With respect especially to dis-

eases of the bladder and urethra, the observations in this book first threw light on those affections and formed the basis on which our present accurate knowledge of them has been founded."

Compared with this part of the book, the chapters on the strictly venereal conditions have at times a certain vagueness of expression and are less positive in their statements. Again we have recourse to Babbington, for he sums this up shortly and truly.

He tells us that: "In the venereal disease we have an abundance of facts, of a certain kind of record. It is not additional experience that we want, but a more correct discrimination, which would enable us better to understand our experience, to disentangle our perplexities, and

Let us dip into the book and look for the new positive and outstanding facts which a physician of the times could learn from its perusal, giving in many cases leads to better treatment.

Although Marcellus Cumanus had given a description of indurated venereal sores in 1495, and John of Vigo, about the same time, had also described them, it was not until Hunter's study that the lesion was clearly differentiated as a venereal entity, whose appearance we know now is followed sooner or later by the symptoms of constitutional syphilis. A great medical historian says: "He differentiated clearly between the hard (Hunterian) chancre and the chancroid ulcer, but his auto-inoculation seems to have confused gonorrhoea and syphilis, a confusion which obtained until the time of Ricord." . . . "He accidentally inoculated himself with lues and purposely delayed treatment in order to study the disease in his own person."

There it is, the fateful May, 1767; we cannot get away from it, and the unfortunate conclusions drawn from the results. According to Hunter's belief, the two affections were due to one and the same virus, sometimes producing one disease, sometimes the other. We know that as a result of what happened, Hunter put off his marriage to Miss Home for three years, and he was not a young man then. Was the inoculation of syphilis accidental? Was Hunter sure, according to his theories, that the other action of the virus might not occur to him,—a possibility which he has reckoned with and was willing to pay the cost of, for the sake of science and humanity?

Soon after this time there was a mania for inoculation experiments, to prove or disprove the theory of the common cause of the two diseases. Physicians inoculated themselves, patients, monkeys, fowls, rabbits, pigeons, and guinea pigs. Medical students experimented personally along the same lines, one physician in France inoculating himself some three thousand times. Hunter tells us that in 1753 it was supposed that the discharge in gonorrhoea came from one or more ulcerations within the urethra. Soon after this the falsity of this doctrine was proved by him. Certain autopsy findings had proved to William Hunter that pus could be formed without any ulceration and loss of substance. This fact was written of by William Sharp, who obtained the knowledge from William Hunter. Sharp and Gatacre wrote of it in connection with other inflammation, Gatacre with special reference to gonorrhoea, having obtained his knowledge from John Hunter's dissections. In the spring of 1753, eight men were executed in Lodon; two of them Hunter knew were affected with the disease in an acute form. He obtained the bodies for investigation, and by dissection and examination of the urethrae proved that there was no ulceration whatever, —only a little more redness and congestion than natural. After this, Hunter gave ceaseless attention to the subject and dissected many urethrae of men affected with the trouble, never finding any indication of any ulceration of any sort.

On the treatment of gonorrhoea by purgation, he says: "On the supposition of the cure being promoted by an evacuation from the blood, what service can purging out some of the blood in form of a secretion from one part do, to an inflammation of another part? On such a supposition would not a sweat, or an increase of saliva from chewing tobacco, or stimulating the nose by snuff all tend equally to cure this trouble?"

The treatment of organic strictures is given fully with the history of the use of the bougie. When he first started in practice, he tells us that pieces of lead and small candles were used for dilatation. He made great improvements in the type and shape of bougies in later use, and was a pioneer in the subject.

Heroic dosage was given for local uses of different drugs, and their use internally was also in astounding dosage. Locally, two grains of corrosive sublimate in eight ounces of water as an injection, and for the lues venerea internally "a grain of this medicine dissolved in about an ounce of some fluid is generally the dose, and increased according as it agrees with the bowels and according to its effects on the mouth and disease." The same statement appears in every edition.

As the two diseases were thought to have a common origin, buboes and all ulcerations were treated by mercury, most often by the inunction method, and drained where necessity demanded it.

Let us consider very quickly a little of what Hunter says of different bladder conditions, especially with relation to the prostate, and his recognition of the great value of sugar in the treatment of those depleted by a long course of mercury. His description of the middle lobe of the prostate and signs of obstruction from it is classic to this day, published in 1786: "Independent of the enlargement of the lateral lobes of the prostate, a little part of this gland which is situated directly back of the very most posterior part of the urethra becomes enlarged . . . . and making a sort of cone deep in the bladder plays the rôle of a valve at the internal

orifice of the urethra." "One can see this enlargement in the subject, even when it is not very big, in exploring the internal orifice of the urethra through the cavity of the bladder." "This portion of the prostate sometimes grows to form a tumor which projects into the bladder for some distance. This projection proves an obstacle to the introduction of sounds into the bladder and other instruments. The sound must be given a greater curve for this part of the urethra."

An actual illustration is given of a death due to prostatic disease which was accompanied by stone in the bladder. Besides the enlargement of the prostate with its local pressure, the enlarged thickened bladder is described, and the prostatic curve for the end of the catheter is insisted on for proper introduction.

"It sometimes happens," he said, "in cases of swelled prostate gland that the catheter cannot be passed without the utmost difficulty and when this has been the case I have left it in the bladder for fear of not being able to pass it again, and continued it there until the bladder has sufficiently recovered its tone." "It sometimes happens that the irritation from the distension of the bladder and the difficulty of throwing out its contents is so great that the urine is prevented from flowing freely into that viscus from the ureters, which become thereby preternaturally distended. The pelvis of the kidney and the infundibula are also enlarged, but how far this dilatation of the ureters and pelvis is really owing to a mechanical cause, I am not clear. In some cases of long standing, where the bladder was become very thick and has been for a long time acting with great violence, it has affected the mammillae so that the surfaces of these processes produce a matter and perhaps affect even the secreting organs of the kidneys, so that the urine secreted was accompanied with pus. The urine in the above cases is generally stale, before it is thrown out of the bladder." "I have seen the muscular coats of the bladder near half an inch thick and the fasciculi so strong as to form ridges on the inside of the cavity, and I also have seen the fasciculi very thin and even wanting in some parts of the bladder, so that a hernia of the internal coat has taken place between the fasciculi and formed pouches, these pouches arise from the thin parts, not being able to support, as happens in ruptures at the navel or rings of the abdomen."

Hunter noted with others the debility ensuing in his patients after a long course of mercury, and had observed from his army service in the sugar isles that "all the negroes, horses and cattle became extremely lusty and fat in the sugar cane season." And also that "the honey bee swarms live all winter on a few pounds of honey, so we must allow that sugar contains perhaps more real nourishment than any other known substance." He began giving large quantities of sugar to these patients with great benefit to their nutrition.

Why Hunter has so little to say of congenital syphilis, and that in a negative way, is hard to understand, as well as his silence concerning visceral syphilis, which Morgagni had so well described. These phases of the malady were also written of extensively by Astruc, the great French syphilologist, but for some reason he touches on their work but lightly, if at all.

It may seem that in this brief and imperfect sketch of one of three greatest surgeons that the world has ever seen, undue stress has been laid on the purely urologic aspects of the Treatise, but this has been done purposely, as in these chapters Hunter's greatness seems to me exemplified. Of this part of the book we could state with truth that much of it could appear in the journals of the last twenty years, as the thoughts of leading urologists of today on certain of these problems. John Hunter was a urologist a hundred and fifty years ahead of his times.

DR. CUSHING—We have come to know John Hunter particularly well in view of the official and pious orations that are delivered each year in his memory at the Royal College of Surgeons in England. In a recent Hunterian oration, Sir D'Arcy Power who entitles his essay "A Martyr to Science" enters into a detailed discussion of Hunter's self-imposed malady. Hunter of course supposed himself well cured, but the terminal ravages of lues in those days were not understood. Jenner is said to have first diagnosed his later symptoms as those of angina pectoris; and the story of his tragic death at the board meeting in St. George's Hospital is only too well known. Hunter had taken careful notes of his symptoms from the beginning, often dictated to one of his helpers when he himself was too ill to write. As Power says, it never seems to have occurred to him to associate any part of his ill health with the inoculation experiment of 1767. Indeed, Hunter says expressly:

> "It would appear that some parts of the body are much less susceptible of the lues venerea than others; and, not only so, but many parts, so far as we know, are not susceptible of it at all. For we have not yet had every part of the body affected; we have not seen the brain affected, the heart, stomach, liver, kidneys, nor other viscera, although some cases are described in authors."

Hunter as we have seen was self taught and he apparently did not read at all thoroughly even the works of his contemporaries; he was the type who preferred to take his facts straight from the book of life rather than second hand. This may have its advantages as well as its disadvantages. His ideas are not often expressed with sufficient clearness for us readily to grasp them. This is particularly true of the last of his four major treatises, *On the Blood Inflammation and Gunshot Wounds*. The work was unfinished at the time of Hunter's death and was posthumously published by his brother-in-law, Everard Home. We shall hear something of

Hunter's studies on the blood from the next speaker.

DR. BOCK—Hunter's observations on the blood were made both in vivo and in vitro. He observed the flow of blood through the capillaries of living animals, noted the phenomena of blood flow through vessels during surgical procedures in human beings, studied the influence of respiration on the appearance of the blood, and carried out a long series of experiments in an endeavor to understand the process of coagulation. His knowledge of comparative biology is apparent throughout the Treatise but this appears to the modern reader to be the source of confusion in his mind as to the real importance of the red corpuscles. No one can fail to be impressed with the multiplicity of experiments designed to uncover the secrets of the blood, done by a man who delved deeply into the field of comparative anatomy and biology, medicine and surgery.

"Blood is most probably as much alike in all animals as the muscle of one animal is like that of another; only with this difference, that some animals have not that part which gives it the red colour; but the other parts, as the lymph and serum, are, as far as I yet know, the same in all."

Hunter conceived that more was to be learned about the use of blood from its coagulation than from its fluidity and the Treatise therefore begins with the subject of coagulation. The process of coagulation divides the blood into three parts: coaguable lymph, serum, and the red globules. During coagulation the red globules become entangled in the coaguable lymph. "The first stage in coagulation is a species of decomposition, forming a separation of the serum." He thought, however, that serum and coaguable lymph might exist separately even in the fluid state of the blood. He noted that the circumstances attending coagulation varied, depending upon the state of the body at the time, and also that the character of the clot varied in firmness or looseness. The three parts were found to vary in gravity, the serum being the lightest, the red globules the heaviest.

The time taken for bleeding and the amount drawn influenced the speed of coagulation, as did also the character of the surface to which blood was exposed, whether that of a dish or an animal membrane, or in the blood vessel itself. A good description of the appearance of the serum and the clot is given, together with an account of the contraction of the clot on standing. The usual reasons given for clotting of blood, such as heat, cold, contact with air, and rest, Hunter believed to be ill founded. Having observed a great variety of conditions under which blood may or may not coagulate, he reached the following conclusion:

"My opinion is that it coagulates from an impression; that is, its fluidity, under such circumstances, being improper, or no longer necessary, it coagulates to answer now the necessary purpose of solidity . . . For I have reason to believe that blood has the power of action within itself according to the stimulus of necessity. . . ."

"The coagulating lymph of the blood being common to all animals, while the red particles are not, we must suppose it from this alone, to be the most essential part: and as we find it capable of undergoing in certain circumstances, spontaneous changes, which are necessary to the growth, continuance and preservation of the animal; while to the other parts we cannot assign any such uses, we have still more reason to suppose it the most essential part of the blood in every animal."

"The serum is the second part of which the whole mass of blood appears to be composed. . . Serum, I believe, is common to the blood of all animals; but there is more of it, I think, in those animals which have red blood; perhaps it may bear some proportion to the quantity of red particles in the blood, and may be of use to dilute it. . . The serum is commonly of a yellowish colour, sometimes more so than at others; and this I should conceive arises from the substances dissolved in it by means of the water it contains." Serum consists of a coaguable and noncoaguable part. The coaguable part was found to be influenced by heat, spirits of wine, volatile spirits, salt of hartshorn, etc. Other body fluids were compared with the reactions of serum, such as water taken from the leg of a dropsical boy, ascitic fluid, and liquor amnii. "The use of serum is probably to keep suspended, and undissolved, the red globules. . . It is also intended to suspend and dissolve any foreign substances in the blood, whether they are of use to the body or otherwise, acting upon them as a common solvent. Thus we see in a jaundiced person, the serum is yellower than common. . . It is probably the solvent of all our secretions."

"The red part of the blood I choose to consider last, although it has been more the object of attention than the other two, because I believe it to be the least important; for it is not an universal ingredient in the blood of animals, like the coagulating lymph, and the serum."

Knowledge concerning the appearance of red corpuscles Hunter notes as of recent date, since the invention of magnifying glasses.

"Malpighi was probably the first who employed the microscope for this purpose, and he, in 1668, wrote a description of the appearance of the globules in the blood vessels of the omentum, which he mistook, however, for globules of fat. Microscopical observations were pursued with great ardour by Antonious Van Leeuwenhoeck, who saw the red globules, August 15th, 1663. These early observers probably imagined more than they saw." (Hunter distrusted the accuracy of magnifying glasses.) "The red globules are nearly always of the same size in the same animals. . . They really have a determined shape and size. . . What this property in the red part is, I do not know, for it has something of the nature of a solid body, yet the particles seem not to have the properties of a solid; for to the touch they yield no feeling of solidity; when circulating in the vessels they may be seen to assume elliptical forms, adapting themselves to the size of the vessels; they must, therefore, be a fluid, with an attraction to themselves while in the serum, which forms them into round globules, yet without the power of uniting with one another, which may arise from their central attraction extending no further than their circumferences; if they are found, however, of an oval figure in some animals, as authors have described, that circumstance would rather oppose the idea of their being a fluid having a central attraction; but this is probably an optical deception (Cf Hewson). Whatever their shape is, I should suppose it to be always the same, in the same animals, and, indeed, in all animals, as it must depend upon a fixed principle in the globule itself."

In the circulation Hunter described the changing color and shape of the red corpuscles as well

as the different velocities with which they passed along vessels in different parts. Solubility tests were carried out with many substances.

As a result of many observations and experiments Hunter concludes:

"The red globules certainly are not of such universal use as the coagulating lymph, since they are not to be found in all animals, nor so early in those that have them, nor are they pushed into the extreme arteries where we must suppose the coagulating lymph reaches; neither do they appear to be so readily formed. This being the case, we must conclude them not to be the important part of the blood, in contributing to growth, repair, etc. Their use would seem to be connected with strength; for the stronger the animal the more it has of the red globules; and the strength acquired by exercise increases their proportion; not only in the whole body, but, as we shall find, occasions them to be carried into parts where in either a quiet or debilitated state of the animal they were not allowed to go: the use, therefore, of a part, and the quantity of red globules passing through it are probably well proportioned to each other."

Hunter believed the color of different parts of the body was determined by the amount of blood in them. White muscle he thought had smaller vessels in it than red muscle and was therefore deficient in red globules.

"Many circumstances in life increase the quantity of red globules, or make them more universal in the muscles of the same animal: thus exercise increases the quantity of the red globules, and the red colour of muscles, while there is the same quantity on the whole, or perhaps we should rather say, that indolence increases the quantity; this is particularly remarkable in women; and probably the whiteness of young animals may arise from the same cause: I suspect, however, something more; I conceive it arises from the principle of life influenced by accidental or mechanical causes."

Many pages of the Treatise deal with the florid color of the blood in the arteries and the purple color in the veins.

"Many substances change the colour of the blood from the modena (or purple) to the scarlet; respirable air has this effect, and many of the neutral salts, more especially nitre, which occasions the florid color in meat which has been salted, also with sea salt. But, as the air produces this effect in the living body, and as we find that without air the animal dies, great stress has been laid on the change of colour, whereas, it should only be considered as a sign that the blood has been in contact with the air: but not that it must be for the purpose of circulation. This effect takes place readily under many circumstances; it takes place out of the circulation as readily as within it; as readily when blood is coagulated as before; it takes place in blood whose coagulating principle has been destroyed, as by lightning, sudden death, etc.: it does not, therefore, depend upon life. It is the cause only of this change in colour by respirable air, which becomes an object of consideration, for if we suppose the change in color in the red globules to be all that respiration is to perform, we shall make the red globules the most essential part of the blood, whereas they are the least so. Most probably the effect of air upon the blood is greatest on the coagulating lymph; and this conjecture is rendered more likely when we consider that in animals which have no red globules of any kind, respiration is as essential to their existence as in any other."

Through the invention of a pair of double bellows, Hunter was able with the aid of artificial respiration to show experimentally that the action of air on the blood in the lungs produced the change in color from purple to scarlet. The effect of irregularities in breathing in patients was studied by observing the changes in color of blood allowed to flow from the temporal artery. Cardiac asthma in a case of rheumatic heart disease and the study of a boy with congenital heart disease also afforded the opportunity to study the color of the arterial blood.

Had the knowledge not been forgotten of that great English school of a century before Hunter, composed of Robert Boyle, Robert Hooke, Richard Lower and John Mayow, Hunter might have spared himself many a tedious experiment. For Boyle had shown that air was essential to life, Hooke invented an apparatus for artificial respiration, Lower carried on blood transfusions and also showed beyond a doubt that the change in color of blood was due to contact with air in the lungs. Mayow showed that only a certain part of the air was responsible for this change. Stephen Hales, at about the time of Hunter's birth, actually attempted an analysis of the air. The work of Hunter's own contemporaries, Black, Rutherford, Priestly and Lavoisier was apparently not known to him. The wonder is not that he failed to interpret correctly the changes wrought on the blood by the lungs but that his inquisitive mind and insatiable thirst for knowledge led him to pursue the problem to the very door the more scientific of his brethren were opening at the time he worked.

The Treatise on the blood closes with a discussion of the living principle of the blood. That the blood had life Hunter had believed for thirty years.

"To conceive that blood is endowed with life while circulating is perhaps carrying the imagination as far as it well can go; but the difficulty arises merely from it being fluid, the mind not being accustomed to the idea of a living fluid. . . When all the circumstances attending this fluid are fully considered, the idea that it has life within itself may not appear so difficult to comprehend: . . . When we consider that every part is formed from the blood, that we grow out of it, and if it has not life previous to this operation, it must then acquire it in the act of forming; for we all give our assent to the existence of life in the parts when once formed. Our ideas of life, have been so much connected with organic bodies, and principally those endowed with visible action, that it requires a new bend to the mind to make it conceive that these circumstances are not inseparable."

Much experimental data is presented to substantiate the hypothesis of a living principle in blood, but time does not permit an account of these. Hunter was an objective worker with a descriptive rather than analytical turn of mind. He spoke of the body as a machine but, as his views on the blood indicate, his philosophy was not that of a mechanist. Had he had the advantages of early training in physics and chemistry, it is possible that knowledge of these

sciences might have greatly aided him in the interpretation of his observations on the blood. It is conceivable, however, that such training might have limited his field of inquiry and prevented his fruitful attack on many other fields of knowledge. In these days of increasing specialization in medicine in ever narrowing lines man himself, as a patient, is in danger of being forgotten because of the emphasis placed on this or that system isolated from his machine as a whole. Sooner or later, some of us as physicians must sacrifice the last word in knowledge in particular fields for the Hunterian outlook.

DR. CUSHING—The five elaborate treatises of which we have heard, and which were issued in the form of these fat quartos exhibited here, represent Hunter's chief publications. They must be taken, however, only as samples of his work much of which has come down to us in most fragmentary form. The story of how his brother-in-law finally destroyed not only the notes which described the specimens in the collection but the mass of documents of other sorts has been told in the biographies. Fortunately the faithful Clift copied out many of the MSS before they fell into Home's hands and Richard Owen, Clift's son-in-law, subsequently published them.

Among the accessories shown in the familiar portrait of Hunter by Reynolds there appear a number of objects which Hunter in all probability selected himself, and which may therefore be taken to represent what, at the time the portrait was painted, he considered to be his most important discoveries. The legs of the skeleton of the Irish giant shown in the background indicate his great interest in the general principles responsible for growth. On the table are two books, one of them labelled "Natural History of Vegetables" and the other "Natural History of Fossils". Under a bell-jar is a rare pathological specimen representing what are said to be multiple bony sarcomatous metastases to the lungs. Standing open on the table is a folio volume on one page of which is shown a graded series of skulls in a descending scale from man to monkey to dog to crocodile, and on the other a graded series of skeletal forelimbs from that of the horse through dog to monkey to man. What Hunter's notes had to say about these pre-Darwinian gradations of ascent and descent we shall never know; but these accessories to Reynolds' portrait are enough to show that even fully to round out an evening devoted to Hunter we would have to call in a botanist, a geologist, a zoologist, a comparative physiologist, an evolutionist, and pathologist.

There is one interest, as already mentioned, that was common alike to Harvey, Malpighi, and to Hunter: namely, the subject of generation. Dr. Lewis will speak to us concerning Hunter's embryological studies.

DR. LEWIS—Hunter's embryology suggests the work of Eustachius in anatomy, which remained unpublished—*in tenebris*—until the day of its usefulness had departed. There is this difference. In a little volume of Opuscula which gleams from the dust of the 16th century, Eustachius published the best that he had done; Hunter published only three pictures of chick embryos, appended to his treatise on "Blood, Inflammation and Gunshot Wounds," with a faulty description generally attributed to the editor. Hunter used these pictures of embryos in support of two propositions:—first, that "the red part of the blood" is "the least important," since in the chick "we see the heart beating when it contains a transparent fluid before any red blood globules are formed;" second, that "new blood vessels are not always elongations from the original ones, but vessels newly formed, which afterwards open a communication with the original." Both of these are doubtful propositions, when applied to the adult, and it was a futile attempt to guide the practice of surgery by the study of avian blastoderms. There will always be better reasons for studying embryology than its relation to medical practice.

What then did Hunter accomplish? He studied the development of eggs for fifteen years—from 1775 to 1790. "Surely one might suppose this the great work of his life," comments Sir D'Arcy Power, "yet it seems to have been a casual by-the-way pursuit."

Previously embryologists had used hens, and Hunter notes that their mode of propagation would almost appear "intended for investigation." "But," he continues, "I found the first appearances so obscure, from want of size in the object, that I had recourse to the Goose. I attempted the Swan, but it was impossible to procure the requisite numbers. I endeavoured to procure ostrich's eggs, but getting only one or two in thirty years, nothing could be made of them. So I kept a flock of geese for more than fifteen years."

William Bell, anatomist and artist, though "an uneducated man," lived with Hunter throughout this period and drew the goose embryos. Sixty of these drawings, engraved by an artist of repute, James Basire, were published by Owen in 1841, together with all of Hunter's brief manuscript concerning them.[1] Nothing on this subject had been burned by Sir Everard.

1 John Hunter's Observations on Animal Development edited, and his illustrations of that process in the bird described, by Richard Owen, F.R.S. London: printed by R. and J. E. Taylor, Red Lion Court, Fleet Street, 1841. Folio, 64 pp., 12 plates (numbered LXVII-LXXVIII). This publication is apparently detached from the 'Catalogue of the physiological series of comparative anatomy contained in the museum of the Royal College of Surgeons in London', vol. 5, 4to, London, 1840. The main text is reprinted in Hunter's 'Essays and Observations', vol. 1, London, 1861, p. 199-216, followed by many of Hunter's 'loose notes and queries on generation'. It is not in the 'Works', London, 1837.

In describing the unfertilized egg, Hunter criticizes the term 'albumen,' which means white, since it is not white until coagulated, and prefers to call it 'the slime.' The rotation of the yolk, so that the cicatricula is always on top, Hunter correctly ascribes to one side of the yolk being lighter than the other, "which side always keeps uppermost, let the egg be ever so often turned."

To examine the embryos, Hunter broke a hole in the shell the size of a shilling, placed the egg under tepid water, and removed the vitelline membrane. Sometimes, under the membranous embryo, he slipped a piece of thin black ivory and put the whole into spirits, thus observing it against a black background.

The earliest stage which Hunter recorded is of six hours' incubation. There is an axial structure in the midst of concentric halos much as Malpighi had previously shown for the hen at six hours. Malpighi, influenced by his discovery of little plants within seeds, decided that the axial structure was a little chick and declared for preformation. Hunter is more wary. "All our observations can only begin at a visible stage of formation, prior to which we are left to conjecture, which could only lead us back to still fewer parts; but when the first and necessary parts were first formed. . . is not known, nor can it."[2]

2 *l. c.*, p. 14. This is preceded by an incredibly awkward statement of the parallelism existing between the embryonic stages of higher animals and the adult condition of lower animals. Foreshadowed by Aristotle, this philosophical idea became the subject of a famous essay by Meckel in 1811, and was thereafter the central theme of embryologists. Hunter 'penned the following passage:'—"If we are capable of following the progress of increase of the number of the parts of the most perfect animal, as they first formed in succession, from the very first to its state of full perfection, we should probably be able to compare it with some one of the incomplete animals themselves, of every order of animals in the Creation, being at no stage different from some of the inferior orders; or, in other words, if we were to take a series of animals, from the more imperfect to the perfect, we should probably find an imperfect animal, corresponding with some stage of the most perfect." Owen comments,—"We may perceive from the evident difficulty with which Hunter expresses the idea, that his mind was oppressed with both its novelty and vastness".

The gosling of perhaps eighteen hours shows clearly the undescribed primitive streak,—a detail for a much later day. "The first parts that are visibly formed may be said to be the brain and spinal marrow," Hunter wrote, noting that a transparent and invisible heart may be 'co-existing,' and he adds that "every part is formed on the outside of the animal." This being true of the cord, he finds that a "proper membrane also begins to form to cover it." The membrane in question is the amnion. There is no suggestion that the cord arises as a thickening of the outer layer, nor is there any reference to the earlier work of Malpighi on the same subject. The ugly scratches, made by Malpighi himself, must ever be counted more significant, in respect to the development of the nervous system, than the finished work of Hunter's artists.

"As the chick grows, it presses down the middle of the yolk, first making a deep indent in it." Hunter's figures show, for the first time, that the head can artificially be thrown back out of the fossula which it excavates in the yolk (fig. 1). In this specimen, a hair has been

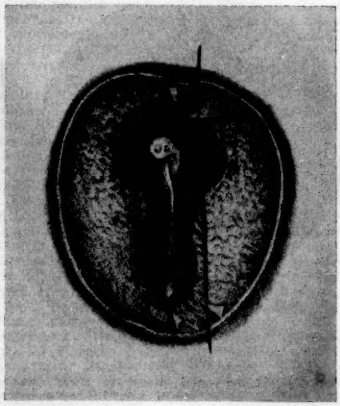


FIG. 1—An embryo goose, drawn for Hunter by his artist William Bell, published and labeled by Owen, in "John Hunter's Observations", Pl. LXIX, fig. 7.

inserted under the outermost layer of the embryo, demonstrating, as Owen states, in a clear and unequivocal manner, that the serous layer is distinct from the vascular layer. Hunter makes no comment on this clever manipulation, but he stood on the brink, and might have slipped into a position (as a medical historian describes it) of 'intolerant central glory.' Fate saved him from this plight, and it remained for Pander and von Baer to 'found' embryology on its germ-layer basis. Hunter indeed lifted the ectoderm from a vascular layer, but he did not recognize the splitting of the mesoderm, and of the entoderm he knew less than his predecessor, Wolff.

Of the vascular system, Hunter describes briefly the largest vessels. Malpighi had shown most clearly the aortic arches, and had drawn, as best he could, all the vessels of the blastoderm. The same are shown, with some new details, in the delicate illustrations made for Hunter. Unfortunately an occasional error in the vascular connections suggests the criticism that "a single small calf with its tail on the wrong end would work havoc in an otherwise faultless and charming picture."

Meanwhile, uninfluenced by Hunter's unpublished work, embryology progressed, and masterly pictures of these early stages of the chick were drawn for Pander by his artist Herr d'Alton.[3] But nothing in Pander is more beautiful or accurate than this of Hunter's (fig. 2),

3 Beiträge zur Entwickelungsgeschichte des Hühnchens im Eye, von Dr. Pander. Würzburg, 1817. Folio, IV + 42 pp., 10 pls.

showing the vitelline membrane rolled to one side, the chorion reflected and the splanchnopleure divided, disclosing the yolk beneath. The amnion is unopened; the small allantois is seen.

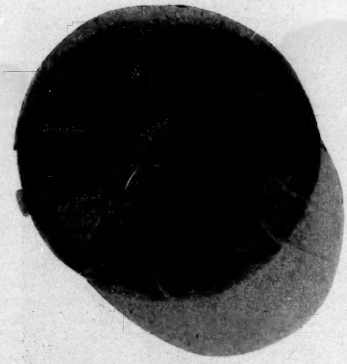


FIGURE 2.

FIG. 2—John Hunter's Observations, Pl. LXXI. The fine engraving of this 'beautiful drawing' (Owen) is hardly suggested by the reduced half-tone.

Probably at the present time there is no simpler or clearer presentation of the three membranous sacs—amniotic, vitelline, and allantoic—than Hunter's drawing (fig. 3), in

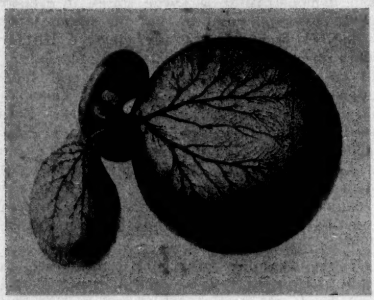


FIG. 3—Side view of an embryo goose showing "the amnios, vitellicle and allantois". John Hunter's Observations, Pl. LXXIV, fig. 3.

which also the chorioid fissure is not to be overlooked. He made several dissections of the digestive tube with the yolk-sac still adherent, but there is no reference to Wolff's earlier demonstration that this tube comes from fore and aft outgrowths of the yolk-sac. Hunter apparently was unaware of that fact. And though Walter Needham, in 1667, had described the yolk-stalk, or *ductus intestinalis*, of birds as forming an *intestinulum quoddam caecum* always present throughout life, and Hunter knew from his own human dissections, of the occasional "appendix or process . . . I believe always from the ileum," it was left for Meckel to unite these observations and forever link his name with the *diverticulum ilei*. Not with impunity could the boy scoff at 'stuffing Latin at the University', for these works, so important for him, were all in Latin.

Malpighi had been greatly impressed with the heart in the chick, which he drew three times, all alike (fig. 4). Blood enters a common auricle, *K*. Thence it flows into a right ventricle, *M*, (on the left of the body!), thence into a left ventricle, *N*, recognized as such since the aorta arises from it. What could one make out of that? Malpighi's pictures, however, are correct. Hunter was apparently on the way to a solution (fig. 5). In too simple a manner he shows the longitudinal division of the bent ventricular limb into right and left portions. But it is made clear that Malpighi's right ventricle includes both ventricles; and his left ventricle is the truncus arteriosus. Hunter did not describe his interesting drawings, which are convincing evidence that he knew more of the development of the heart than Haller, who wrote a book about it in 1758, determined to solve the mystery of Malpighi's figures. But Haller could not do so, and is overcome by 'la beauté parfaite' of the yolk and vessels. "Quelle abondance, et quelle prodigalité d'ornemens pour des êtres si momentanés!" he exclaims, followed by his own inferior version of the Japanese proverb—"the morning-glory envies not the pine of a thousand years."[4] These are among his 'corollaires mêlés.'

FIG. 4—The heart of the chick as drawn by Malpighi in 1672, —De ovo incubato, fig. 29. Other drawings show the aortic arches.

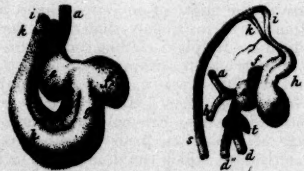


FIG. 5—Fundamental features of the development of the chambers of the heart, first clearly shown in the unlabeled drawings for John Hunter. Observations, Pl. LXXVI, figs. 7 and 8.

4 Sur la formation du coeur dans le poulet; etc., par Mr. de Haller. Lausanne, 1758. 2 vols., 16mo. 472 and 368 pp. Mém. II, p. 191,—Sa sagesse créatrice orne aussi bien le champignon, qui ne dure que deux jours, que le chene, qui a des siecles à vivre.

A prominent feature of several of Hunter's figures of chicks toward the time of hatching is the little horny knob, or shell-breaking process, on the upper mandible. Aldrovandi had written of it in 1599, thinking that it acted like a

button on a fencing foil to prevent rupture of the membranes by the sharp beak. Women, he said, say that chickens cannot pick up corn until it has been shed. Sharp as a pin, this spine is used to break through the shell, and the chick then turns to the left, detaching a round cap from the larger end, which may be inverted and fitted into the rest of the shell in the neatest possible manner. Yarrell described the appendage as a new discovery in 1826. There are still no better figures of its external form than those of Hunter.

The ruptor of the shell is scarcely indicated in the picture (fig. 6), with which Hunter con-

FIG. 6—John Hunter's Observations, Pl. LXXVIII,—a gosling near the close of incubation.

cludes his series. This forlorn bird seems to ask of the whole undertaking,— *Cui bono?* Had this atlas been published in 1793, with text commensurate with the figures, and with due correlation with previous literature, it might have been a work in embryology second to none. "The last of Hunter's scientific occupations, and in which Mr. Clift was assisting the evening before his master's sudden death, was the arrangement of the drawings, preparatory to their description."

DR. CUSHING—Among Hunter's many activities, there is one that seems to have been largely lost sight of: namely, his interest in entomology. I have long wished to know what someone thoroughly versed in the subject would have to say about Hunter's studies in this direction. Among the Hunteriana displayed there is a letter which may appropriately be read before introducing the next speaker.

(Leister Square, Feby. 2d, 1792.
To Sir Joseph Banks, Brt. P. R. S.)

Sir

Allow me to present to you a Paper on the natural history of the common Bee. It contains the result of experiments and observations, occasionally made in the course of the last twenty years. They have been frequently interrupted by my other pursuits, which prevented me from following this subject regularly through any one season, and consequently obliged me to renew it in others. Unforseen accidents have likewise considerably retarded my progress; a very sultry day melting the comb; the Bees removing some eggs or maggots; or a Hive dying under experiment, have destroyed the chain of my observations for a whole season.

It was my intention to have added some remarks on the management of Bees in this country, but I am induced from the length of this paper to reserve them for another opportunity. If you and your learned council think them worthy the attention of the Royal Society, I shall hope to have them honoured by a place in your valuable transactions.

As I have gone a considerable length into the natural history of this tribe of Insects, I hope next winter to give you the account of the wasp and probably the Hornet. I shall afterwards be able to give the wild or Humble Bee, and many of those called solitary Bees, in which I have made considerable progress, and in time I hope to complete the history of the British Bees.

I am Sir your much obliged and most Humble Servant.

JOHN HUNTER.

The paper was read before the Royal Society three weeks later and if anyone can tell us what Hunter's studies on bees and other insects amounted to, I am sure Professor Wheeler can.

PROF. WHEELER—John Hunter did so much on natural history besides all of his medical works that he probably deserves place as the most important naturalist between Aristotle and Darwin.

Dr. Cushing has mentioned a number of his important papers, those on the electric fishes, and also a paper on the Free Martin, those peculiar twins of different sexes in cattle in which the female is influenced by the hormones of the male. Hunter seems to have been the first to have called attention to these peculiar conditions.

On looking over his work on insects we notice that Richard Owen, who edited the papers, had asked Frederick Smith to annotate the papers on insects. That leads me to relate an anecdote which I have heard in regard to Frederick Smith. He and Francis Walker and probably several other entomologists were employed by the British Museum to describe insects. The museum had accumulated a number of insects from the tropics and they were paid a shilling for every new insect they described. It is said that they spent the morning describing species as rapidly as possible, then went out to lunch and when they came back forgot where they left off and described many of them over again,

so that entomologists have been worrying about their specific identity ever since.

Frederick Smith was not the best person to annotate these works because they cover too many biological subjects with which he was not familiar. I wish to mention only one important thing that Hunter discovered in connection with the honey-bee. It is surprising that he could make any discoveries in this insect because two entomologists had studied it very carefully before him. One of these was Swammerdam. He was the first to do anything with honey-bees since Aristotle and discovered that the queen is really a queen and not a king. In his "Biblia Naturae" which I have brought here there is a beautiful picture of the ovaries of the queen which is so fine that it is still used as a text-book figure. There is also in it a picture of the tongue of the honey-bee. Swammerdam made the mistake of regarding the tongue as a hollow tube, whereas it is merely grooved. Of course, he made many other interesting discoveries. Réaumur came along a few years later and devoted nearly the whole of one of the six volumes of his "Mémoirs" to the honey-bee, amplifying and improving on Swammerdam and using very interesting artificial hives. In the first part of the book there are pictures of these hives set up in his garden. In the back is his interpretation of the tongue, showing that it is a grooved organ and not a tube.

Along toward the end of the century Hunter made the discovery to which I refer. Both Swammerdam and Réaumur and all the older writers, of course, knew that the bees brought in pollen as food but they never succeeded in making out where the wax came from. Both Swammerdam and Réaumur believed that it was made out of the pollen, or farina, as Hunter called it. But Hunter really did discover the source of the wax. Strangely enough, the entomologists have never said anything about it, probably because they have not seen the articles of Hunter and the discovery is usually attributed to François Huber, who was a famous French-Swiss entomologist. He was blind and made all his remarkable observations on the honey-bee with the aid of a servant. He also invented a great many new artificial hives. In his book you will see a plate with the secretion of wax between the abdominal segments of the worker bee and also pictures of the hives.

I will read a paragraph in Hunter's paper on the bee, in regard to secretion of the wax. He recognized it as a discovery:

"The materials of their dwelling, or comb, which is the wax, is the next consideration, with the mode of forming, preparing, or disposing of it. In giving a totally new account of the wax, I shall first show it can hardly be what it has been supposed to be: First I shall observe that the materials, as they are found composing the comb, are not to be found in the same state (as a composition) in any vegetable, where they have been supposed to be got. The substance brought in on their legs, which is farina of the flowers of plants, is, in common, I believe, imagined to be the materials of which the wax is made, for it is called by most the wax: but it is the farina, for it is always the same colour as the farina of the flower where they are gathering; and indeed we see them gathering it, and we also see them covered almost all over with it, like a dust; nevertheless, it has been supposed to be the wax, or that the wax was extracted from it. Reaumur is of this opinion. I made several experiments to see if there was such a quantity of oil in it as would account for the quantity of wax to be formed, and to learn if it was composed of oil. I held it near the candle; it burnt, but did not smell like wax, and had the same smell when burning, as farina when it was burnt. I observed that this substance was of different colours on different bees, but always the same colour on both legs of the same bee; whereas new-made comb was all of one colour. I observed that it was gathered with more avidity for old hives, where the comb is complete, than for those hives where it is only begun, which we could hardly conceive if it was the materials of wax: also we may observe that at the very beginning of a hive, the bees seldom bring in any substance on their legs for two or three days, and after that the farina gatherers begin to increase; for now some cells are formed to hold it as a store, and some eggs are laid, which when hatched will require this substance as food, and which will be ready when the weather is wet. I have also observed, that when the weather has either been so cold, or so wet, in June, as to hinder a young swarm from going abroad, they have yet in that time formed as much new comb as they did in the same time when the weather was such as allowed them to go abroad. I have seen them bring it in about the latter end of March, and have observed in glass hives the bees with the farina on their legs, and have seen them disposing of it, as will be described hereafter.

The wax is formed by the bees themselves, it may be called an external secretion of oil, and I have found that it is formed between each scale of the under side of the belly. When I first observed this substance, in my examination of the working bee, I was at a loss to say what it was: I asked myself if it was new scales forming, and whether they cast their old, as the lobster, &c. does? But it was to be found only between the scales, on the lower side of the belly."

Hunter then goes on to say that he took some of it on a needle and melted it and found it was the wax, so I think we should put that down as an important discovery since those two eminent naturalists, Swammerdam and Réaumur had quite failed to notice the way in which wax is secreted.

There was one other interesting matter that I noticed in one of the volumes and that is a short paper on the social instincts. Even at that time when Rousseau and others believed that human society was due to a contact between different individuals, Hunter believed that the association of human beings and of animals that form societies is rather an appetite. He says:

"The hornet, bee, wasp, etc. will fight any animal (perhaps those of their own species more readily) that may intrude on their domestic concerns; but they will not pay attention to one another, when they meet on the same flower, on a ripe peach, etc.

The same principle exists in the Human race: Men have their degrees of attraction towards each other according as they are circumstanced in life. In close connections he is sociable, excepting inter-

ests clash (in which case no animal is sociable). In the crowd he forms his likings, dislikings, and indifferences. But, take him to Siberia, and let him meet the man he most disliked in his own country; he will immediately become sociable with that man. He is, in such a situation, deprived of the acquired cause of sociability, viz. acquaintance; and he feels the want of such; so that the moment the object of acquaintance presents himself, he associates with him; for the dislike arose from having had great choice. This becomes exactly similar to an appetite. A man, in the midst of a great variety of foods, forms his likings and dislikings, which arises from having choice; but, take away that choice, and let him have new kinds of foods which his appetite does not associate with, and he will immediately take to the old food he had been in some degree accustomed to, but which he had disliked.

Upon this principle, animals may be made to associate that otherwise would not. Thus, if we wish a strange animal to associate with others accustomed to a place, take the whole into a strange place, and let them all stay there but one day; and then bring them home, and they will all seem as well as acquainted as if they had been brought up together.

A gentleman gave me a lamb with three legs, which my other sheep had nearly killed; and after repeated trials, the farmer put it into a house. I ordered the whole flock to be driven to a strange field which they had never been in before; and, the moment they were out of their own territories, they allowed the lamb to herd with them; and when brought home in the evening to the old field, they took no more notice of it, at least by way of hostility.

I thought that was very interesting because it is a view that has been stressed quite recently by some psychologists. The animal, for instance, that is removed from the herd becomes very uneasy until it gets back in among the rest of his fellows and is then quite satisfied, a behaviour very much like that which we notice in appetites, so that in this matter Hunter was evidently quite ahead of his time.

DR. CUSHING in closing—Hunter was the first to teach the *Science* of Surgery as Paré two hundred years before had advanced the *Art*. But as has been said, he made his name immortal by the labour of his own hands outside the sphere of his vocation. He did not travel by the ordinary road and it is impossible to measure him by the standards of other men.

IS THE REHABILITATION OF THE ADVANCED SERO-NEGATIVE TABETIC POSSIBLE?

BY LEROY A. LUCE, M.D.*

THE first step toward the rehabilitation of the advanced tabetic is to render the serology permanently negative, by appropriate anti-syphilitic treatment. For this purpose the Swift-Ellis[1] method has, in my own practice, fulfilled every requirement. In cases where this therapy is successful, it is commonly assumed that the syphilitic process is quiescent, or possibly arrested. Unfortunately, the symptoms and disabilities, although somewhat alleviated, do not usually show sufficient improvement and in spite of the success of the treatment they continue to reappear.

The benefits resulting from this treatment are probably due to the destruction of the spirochætes, to the subsidence of inflammation and inflammatory pressure, and to the reactivation of nerve tissue that has escaped degeneration. If such is the case, we are justified in the hope that re-ëducative, electro-therapeutic, or other stimulative methods, such as were in vogue before the discovery of arsphenamine, may prove to possess some power of rehabilitation.

In my own cases I have found no evidence that such measures are anything more than palliative, and the general opinion is that the sero-negative advanced tabetic cannot to any substantial degree be rehabilitated. For example, Tarkington[2] states that when degenerative changes have occurred, it is impossible to give more than palliative relief. Alford[3] says the most that can be done is the permanent arrest of the degenerative process. Stokes and Shaffer[4] show that the late tabetic is expected to continue to carry evidence of the degenerative aspect of the process. O'Leary and Nelson[5] say that for the sero-negative tabetic who still suffers from gastric crises nothing can be done but chordotomy.

In direct contradiction to these statements, I am happy to say that during the years 1921-1925 I had six cases in which the rehabilitation was practically complete. Five of these cases have continued to hold their own. The other case, the oldest of all, has done even better. In my first case, an advanced sero-negative tabetic, my treatment began an attempt to relieve gastric crises. After failing with other electrical methods, I succeeded in discovering a treatment that appeared to have a beneficial effect on the symptoms, and finally on the disabilities. With the five other cases, which were treated in the same manner, equally good results were obtained.

CASE 1. A married woman, 36 years old. She was married in 1905, contracted syphilis in 1906, and was delivered of a stillborn child in 1907. After contracting syphilis she began to suffer from headache, and for many years was never free from it. At times it became excruciating, and lasted from one to four days, accompanied by violent vomiting and by nausea at the sight of food.

In 1910 she became afflicted with an intense pain at the left side of her spine, which occurred several times a day. This gradually spread round the left side of her body, which grew too sensitive to bear touch. From 1910 to 1920 she suffered intermittently

*For record and address of author see "This Week's Issue," page 839.

from headache, great fatigue, lightning pains in her arms, legs, and back, and gastric crises. Her gait became ataxic. In 1913 she developed clitoral crises, and in 1915 paralytic incontinence of urine. Between 1910 and 1920 her weight fell from 130 pounds to 85.

I saw her first in 1920. She was weak, emaciated, and ataxic, with every symptom of a confirmed invalid. She had great difficulty in walking and inability to do any kind of housework.

EXAMINATION. Her gait was ataxic; Romberg's sign, marked; the pupils were unreactive to light, but reactive to distance, the left pupil being 2mm. larger than the right; the knee jerk and ankle jerk were absent; the heel-to-patella test was inaccurate.

SENSATION. The left side of her trunk, from the fourth to the eighth dorsal vertebra, was insensitive to touch, pain, and temperature. The right side, between the same vertebrae, from the spine to the mid-axillary line, was insensitive in the same way, but to a less degree. The ulnar border of both hands was also insensitive to touch, pain, and temperature. Otherwise the examination was negative.

SEROLOGY. The blood Wassermann reaction was positive; the spinal-fluid Wassermann reaction, positive; cell count, 33; colloidal-gold reaction, 123111000.

DIAGNOSIS. Tabes dorsalis.

TREATMENT. The first part of my treatment was by the Swift-Ellis method. Between April and October, 1920, six times, at fairly regular intervals, three or four tenths of a gram of arsphenamine was given intravenously, and from 20 to 30cc. of arsphenamized serum intraspinously. Each administration brought on lightning pains in her legs, and gastric crises with violent vomiting. These symptoms gradually decreased, and disappeared after about three weeks. The next administration did not take place until several days after the subsidence of the symptoms.

With the hope of avoiding these severe reactions the method of treatment was changed to that of Ogilvie[1]. Between December 1920 and September 1921 six administrations were given. Although the gastric crises were as severe as before, the patient suffered less, since the omission of intravenous arsphenamine did away with the attendant nausea and vomiting.

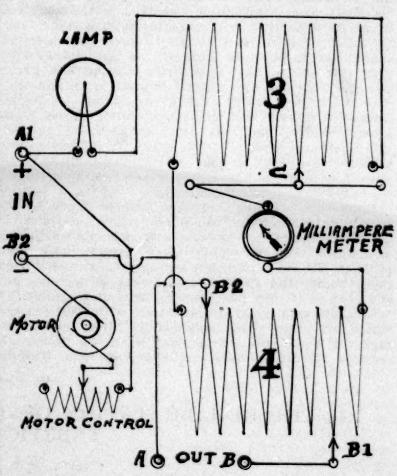
At the end of the twelve administrations the serology of the patient was negative for both the blood and the spinal fluid, and it has remained so ever since. The last test, made in September 1927, showed the blood Wassermann and the spinal fluid Wassermann to be negative. The cell count was four. The colloidal-gold reaction was 000010000. But so far as the patient's rehabilitation was concerned, her condition after these treatments was far from satisfactory, for not only were her symptoms unchanged, but she developed a considerable degree of mental depression.

At this point it was decided to try to relieve her symptoms by the use of electricity. After several months of experimentation, without favorable results, I made use of a different kind of electrical tr atment, herewith described, which I happened upon by accident. The electrical circuit used is shown in the following diagram.

SPECIFICATIONS. A direct current is necessary. It enters at the point marked "A^1 and B^2 IN." The polarity of A^1 is plus; that of B^2 is minus.

To reduce the voltage to the necessary point, the circuit has a 50-Watt carbon lamp. Coil No. 3 consists of 324 turns of No. 28 (Brown & Sharpe gauge) enamel-covered, advance resistance wire (manufactured by the Driver-Harris Company, Harrison, N. J.), wound on a fibre tube 8 in. in length, 3¾ in. in diameter, and completes the coil.

Coil No. 4, the secondary unit, consists of a fibre tube of any reasonable thickness, 4 in. in length and 1 in. in diameter, wound with 150 turns to the inch of nichrome wire (No. 33 Brown & Sharpe gauge). The arrow C is the adjustible slide that controls the current from coil No. 3, and passes through a milliampere-meter to coil No. 4. The arrows B^1 and B^2 are



the oscillating arms that convey the current from the secondary coil No. 4 to the patient, through the points marked "A and B OUT." These oscillating arms move in opposite directions, on opposite sides of the coil, to change the polarity. To produce motion in the arms, it is necessary to use a motor of small design, with a rheostat to control the speed. Any convenient device may be used to connect the motor with the slides. In my experience the best results have been produced by changing the polarity twenty times a minute.

APPLICATION. After a considerable amount of experimentation on Case 1, I adopted the following method, which later experience proved to be the best for general use. With the patient lying on her back on a well-padded table, two electrodes of standard material, four inches wide, and of equal length were applied to her spine from the atlas to the coccyx, the terminals being attached at the remote ends of the electrodes. To produce complete relaxation, the patient lay on the electrodes with her thighs flexed and supported by pillows. At the beginning of each application the current was slowly increased until she became conscious of a metallic taste. At this point the current was gradually diminished until the metallic taste was practically gone, and the current was maintained at this point during the remainder of the application.

The duration of the first application was ten minutes. Subsequently it was varied to fit the patient's condition. The maximum duration was twenty minutes. The dosage ranged from five to forty milliamperes, the average being about thirty. Overstimulation, indicated by the recurrence of lightning pains, with a sensation of stiffness in the legs and of unusual fatigue for twenty-four hours after the treatment, had to be carefully avoided. The application was most effective when made daily. The treatment extended over a period of eight months, Sundays and holidays included.

After the fourth treatment the lightning pains and

gastric crises gradually became less severe, and at the end of six months disappeared. The patient was now able to do her housework without fatigue. Her gait was steady, and she could walk in a straight line forward or backward with her eyes closed. She had no ataxia. She had control of her bladder. The sensory disturbances in her chest were greatly improved. She had regained her knee jerk. There was great improvement in her general condition, and a substantial gain in her weight. The above table indicates her condition before and after the Swift-Ellis and the electrical treatment.

SUMMARY OF THE RESULTS OF TREATMENT—CASE 1

Signs and Symptoms	Before Swift-Ellis Treatment	After Swift-Ellis Treatment	After Electrical Treatment
Romberg's sign	Present	Unchanged	Absent
Knee jerk	Absent	"	Present
Finger-to-nose test	Unsteady and inaccurate	"	Accurate
Heel-to-knee test	Unsteady and inaccurate	"	"
Pupils	Unreactive to light, reactive to distance	"	Unchanged
Lightning pains	Severe	"	Absent
Gastric crises	"	"	"
Explosive vomiting	"	"	"
Clitoral crises	"	"	"
Fatigue on exertion	Acute	"	Normal
Sensation in back	Absent	"	Present, but inconstant and delayed
Sensation in ulnar region of hands	"	"	Present, but inconstant and delayed
Headaches	Severe	"	Absent
Gait	Ataxic	"	Normal
Bladder	Uncontrolled	Slightly controlled	"
General condition	Poor	Unchanged	Excellent
Weight	90 lbs.	85 lbs.	140 lbs.

The patient had had no electrical application for six years. During this period she had been able to carry on the duties of a position requiring both mental and physicial alertness and control. Except for the condition of her pupils she has no symptoms of the tabetic.

CASE 2. A widow, 44 years old, suffering intense abdominal pains, was admitted to a general hospital in September 1920. Her chief complaints, for two years past, were lightning pains in the legs, gastric crises with vomiting, increasing difficulty in walking, double vision, headaches, incontinence of urine, and loss of weight and strength.

EXAMINATION. Her gait was ataxic; Romberg's sign, marked; the pupils irregular in outline and unreactive to light, but reactive to distance; the knee jerk and ankle jerk were absent; the heel-to-patella test, inaccurate. Otherwise the examination was negative. The blood Wassermann and spinal-fluid Wassermann reactions were positive. The colloidal-gold reaction was 123100000.

DIAGNOSIS. Tabes dorsalis, with gastric crises.

TREATMENT. The patient remained in the hospital about five months, and received three Swift-Ellis and five Ogilvie administrations. Her Wassermann reactions during her stay in the hospital were as follows: on October 20, 1920, blood, negative; spinal-fluid, moderately positive. On December 3, 1920, blood, negative; spinal-fluid, slightly positive. When she was discharged from the hospital, her general condition was improved. She was able to eat well and to sleep well; she had no nausea, and no abdominal pains; she still had dribbling urine, unsteady gait, and headaches; the lightning pains in her legs were less severe; her knee jerk and ankle jerk were still absent.

From March to September, 1921, the patient received twelve additional Swift-Ellis administrations. At the end of the treatment her blood Wassermann

SUMMARY OF THE RESULTS OF TREATMENT—CASE 2

Signs and Symptoms	Before Swift-Ellis Treatment	After Swift-Ellis Treatment	After Electrical Treatment
Romberg's sign	Present	Present	Absent
Knee jerk	Absent	Absent	Present
Finger-to-nose test	Unsteady and inaccurate	Unsteady and inaccurate	Accurate
Heel-to-knee test	Unsteady and inaccurate	Unsteady and inaccurate	"
Pupils	Unreactive to light, reactive to distance	Unchanged	Unchanged
Gastric crises	Severe	Absent	Absent
Explosive vomiting	"	"	"
Lightning pains	"	Improved	"
Fatigue on exertion	Acute	Unchanged	Normal
Headaches	Severe	"	Absent
Gait	Ataxic	Improved	Normal
Bladder	Uncontrolled	Slightly controlled	"
General condition	Poor	Improved	Excellent
Weight	128 lbs.	120 lbs.	140 lbs.

and spinal-fluid Wassermann reactions were negative, and had been so for about a month.

Inasmuch as the patient still suffered from lightning pains, lack of bladder control, ataxia, and headaches, I began the electrical treatment as early as possible (on December 1), as described in Case 1, and made on an average three applications a week until June 23, 1923.

The patient was seen yearly from 1924 to 1927. She had maintained all her gains. She had supported herself by running a lodging house, and had done all the necessary work, including the care of the furnace.

Her serology in November 1926 was as follows: blood Wassermann reaction, negative; spinal-fluid Wassermann reaction, negative; cell count, 2; colloidal-gold reaction, 000000000.

CASE 3. A colored male waiter, 61 years old, he had had syphilis for many years. In 1908 he had headaches, pain in his back at the waist, and lightning pains in his legs. In 1914 he was treated at a general hospital for several months, with very little improvement. His condition soon became worse. On June 1, 1921, he suddenly lost the power of locomotion. I saw him first on June 14, with his family doctor. He was not only ataxic, but weak and emaciated. He had general weakness, numbness in his hands and feet, headaches, difficulty in walking, impotence (for four years), loss of bladder control, and lightning pain in his legs; his weight had fallen from 200 pounds to 154.

EXAMINATION. His gait was markedly ataxic; Romberg's sign, pronounced; pupils, unreactive to light, and sluggish to distance; knee jerk and ankle jerk, absent; finger-to-nose test, unsteady and inaccurate; heel-to-patella test, markedly inaccurate; hands and feet showed diminished and delayed sensation of touch, pain, and temperature. Otherwise the examination was negative.

SEROLOGY. The blood Wassermann reaction was negative; the spinal-fluid Wassermann reaction, positive; cell count, 36; colloidal-gold reaction, 112230000.

DIAGNOSIS. Tabes dorsalis.

TREATMENT. The Swift-Ellis method was used, from three to four-tenths of a gram of arsphenamine, and from 25 to 35cc. of arsphenamized serum being given fourteen times, at regular intervals, between June 1921 and February 1922.

At the end of the treatment the patient's serology was as follows: blood Wassermann reaction, negative; spinal-fluid Wassermann reaction, negative; cell count, 4; colloidal-gold reaction, 000110000. Although his serology was negative, he showed, so far as concerned rehabilitation, almost no improvement. For a period of five months I applied electricity daily, as described in Case 1. When the patient was dismissed, in July 1922, he was practically normal.

Six years have passed since the cessation of the electrical treatment. The patient has maintained all his gains. He has a position which requires physical as well as mental alertness. His blood Wassermann and spinal-fluid Wassermann reactions are still negative; his colloidal-gold reaction, 001110000.

CASE 4. An outdoor salesman, 42 years old. In 1910 he had sudden attacks of pain in his stomach, with vomiting, which lasted three days. From 1910 to 1920 these attacks occurred about once a month. In 1911 he developed an unsteady gait, and had great difficulty in walking. In 1918 he found it almost impossible to walk outdoors. In 1919 he began to have difficulty in holding his urine. During the years 1910-1919 he was subject to frequent and severe headaches, to lightning pains in his legs, and to girdle sensation. He had gradually lost his sexual power. He had become incapacitated for any kind of work.

EXAMINATION, September 10, 1920. The exmination showed his gait to be extremely ataxic; Romberg's sign was marked; the pupils were unreactive to light, sluggish to distance, irregular in outline, and unequal, the right pupil being 4mm. larger than the left; the knee jerk and ankle jerk were absent; the abdominal and cremasteric reflexes were present. On the right side of his trunk, from the seventh to the eleventh dorsal verterbra, the sensation of touch pain, and temperature was diminished, delayed and variable; a similar condition of sensation existed in the ulnar border of both hands, and extended halfway to the elbows; it existed in both feet also. The heel-to-patella test, and finger-to-nose test, were unsteady and inaccurate; the blood Wassermann reaction was moderately positive; the spinal-fluid Wassermann reaction, positive; cell count, 120; colloidal-gold reaction 113411100.

DIAGNOSIS. Tabes dorsalis.

TREATMENT. The Swift-Ellis method was employed. From September 14, 1920, to June 7, 1921, from three to four tenths of a gram of arsphenamine and from 25 to 35cc. of arsphenamized serum were administered fourteen times, at regular intervals. At the end of the treatment the patient's serology was negative for his blood and spinal fluid, and has continued to be negative. About six months after the cessation of the treatment his general condition was found to be improved; but although his ataxia was less marked, his bladder control regained, and his headaches less severe, his gastric crises, his

SUMMARY OF THE RESULTS OF TREATMENT—CASE 3

Signs and Symptoms	Before Swift-Ellis Treatment	After Swift-Ellis Treatment	After Electrical Treatment
Romberg's sign	Marked	Slightly improved	Absent
Knee jerk	Absent	Absent	Present
Ankle jerk	"	"	Right, present
Heel-to-patella test	Inaccurate	Unchanged	Accurate
Finger-to-nose test	"	"	"
Pupils	Unreactive to light, sluggish to distance	"	Unchanged
Lightning pains	Severe	Severe	Absent
Fatigue on exertion	Acute	Unchanged	Normal
Sensation in hands and feet	Diminished and delayed	"	"
Headaches	Severe	Unchanged	Absent
Gait	Markedly ataxic	Slightly improved	Normal
Sexual power	Absent	Unchanged	"
Bladder	Uncontrolled	Slightly controlled	"
General condition	Poor	Fair	Excellent
Weight	154 lbs.	154 lbs.	200 lbs.

lightning pains, his sexual power, his sensory disturbances, and his physical incapacitation had remained unchanged. I applied electricity daily, as described in Case 1, for six months.

The patient was seen in September 1924, two years after the cessation of the electrical treatment. During these two years he has been able to resume his occupation as outdoor salesman, and to work full time without undue fatigue. He stated that he had been free from pain, that he had had no bladder trouble, that he was no longer impotent, and that his gait was normal. A test showed that he had no Romberg's sign, and no evidence of superficial sensory disturbances. His general condition was manifestly excellent, as indicated by his substantial gain in weight.

An examination on January 29, 1928, about five and a half years after the cessation of the electrical treatment showed that he had maintained all his gains, and that his general condition was still excellent.

SUMMARY OF THE RESULTS OF TREATMENT—CASE 4

Signs and Symptoms	Before Swift-Ellis Treatment	After Swift-Ellis Treatment	After Electrical Treatment
Romberg's sign	Marked	Unchanged	Absent
Knee jerk	Absent	Absent	"
Ankle jerk	"	"	"
Finger-to-nose test	Unsteady and inaccurate	Unchanged	Accurate
Heel-to-patella test	Unsteady and inaccurate	"	"
Pupils	Unreactive to light, sluggish to distance	"	Unchanged
Gastric crises	Severe	"	Absent
Explosive vomiting	"	"	"
Lightning pains	"	"	"
Fatigue on exertion	Acute	"	Normal
Sensation in back, hands and feet	Diminished and delayed	"	"
Headaches	Severe	Less severe	Absent
Gait	Ataxic	Improved	Normal
Sexual power	Absent	Unchanged	"
Bladder	Dribbling	Normal	"
General condition	Poor	Improved	Excellent
Weight	90 lbs.	95 lbs.	113 lbs. (normal)

Case 5. A colored man, 66 years old, in 1876 had contracted syphilis and received anti-syphilitic treatment for about a month. In 1895 he began to have difficulty in walking or running, or in climbing stairs, and to suffer from lightning pains in his legs. A year later, while working as a waiter, he lost the use of his legs, and had to be carried home. From this time on he was practically an invalid. He walked with great difficulty, and only with the aid of two canes. After another year, during which conditions remained about the same, he was treated at a general hospital, without appreciable benefit. The diagnosis was locomotor ataxia.

In 1909 he was treated at another general hospital for a Charcot's joint. In 1913 he received twelve intravenous arsphenamine injections, with some relief to his lightning pains, but with no improvement in his ataxia.

For the past twenty years he has been unable to stand without assistance. During this long period his gait has been shuffling, on account of his inability to lift his feet, and he has been subject to morning nausea, vertigo, and depression. For the past ten years the lightning pains have been severe enough to keep him awake for several days at a time. For

SUMMARY OF THE RESULTS OF TREATMENT—CASE 5

Signs and Symptoms	Before Electrical Treatment	After Electrical Treatment
Romberg's sign	Marked since 1896	Absent
Knee jerk	Absent	"
Ankle jerk	"	"
Finger-to-nose test	Unsteady and inaccurate	Accurate
Heel-to-patella test	Impossible	Steady and accurate, but unable to touch patella on account of Charcot's joint
Pupils	Unreactive to light, sluggish to distance	Unchanged
Lightning pains	Extreme	Absent
Fatigue on exertion	Marked	Normal
Sensation in hands and feet	Absent to touch, pain, and temperature	Present in hands, but diminished and delayed; absent to touch, pain, and temperature in feet.
Sense of position in toes	Absent	Absent
Morning nausea and vertigo	Daily for twenty years	"
Gait	Impossible without assistance	Normal but for Charcot's joint
Sexual power	Absent for ten years	Normal
Bladder	Uncontrolled	"
General condition	Fair	Good
Weight	140 lbs.	160 lbs.

the past three years he has been impotent, and has dribbled.

An examination on June 10, 1922, showed the patient still unable to maintain his balance without support, or to walk without the aid of canes. His pupils were unreactive to light, myotic, and sluggish to distance. The knee jerk and ankle jerk were absent; the triceps, biceps, abdominal, and cremasteric reflexes were present. In his hands and feet he was insensitive to touch, pain, and temperature. In his toes he had no sense of position. The heel-to-patella test was impossible, since he could not raise either leg from the table. The finger-to-nose test was unsteady and inaccurate, with both hands. He showed no hypotonia or atrophy of muscles.

DIAGNOSIS. Tabes dorsalis, burned out.

Inasmuch as his serology was already negative, I began the electrical treatment at once (on June 28, 1922), and made a daily application until August 31, 1923.

An examination on June 27, 1927, nearly four years after the cessation of the electrical treatment, showed that the patient not only had maintained all his improvement, but had made certain marked gains. In his hands he had recovered the sensation of touch, pain, and temperature. In his feet he could differentiate the point of the pin from the head, and he had recovered the sensation of touch, pain, and the temperature to a degree approaching normal. In his toes the sense of position manifested itself in about half the tests.

CASE 6. An electrician, 33 years old, was admitted on June 4, 1922, to a general hospital. Four days before, while at his work, his left leg collapsed, and he had to be carried home. On the following day he had a similar experience. Two days later he had intense abdominal pains with vomiting, which continued for several hours after his admission into the hospital. For a year he had had occasional attacks of abdominal pains with vomiting, and his gait had become so unsteady that he found walking difficult. Furthermore, he had lightning pains in his legs, he was unable to control his urine and feces, and he was impotent.

EXAMINATION. The patient was unable to walk or to stand without support; the pupils were irregular, and unreactive to light, but reactive to distance, the right pupil being larger than the left; the knee jerk and ankle jerk were absent; the finger-to-nose and heel-to-knee tests, unsteady and inaccurate. His chief complaints were inability to walk, abdominal pains with vomiting, lightning pains in the legs, incontinence of urine and feces, and impotence. The blood Wassermann and spinal-fluid Wassermann reactions were positive; cell count, 43; colloidal-gold reaction, 114441000.

DIAGNOSIS. Tabes dorsalis, with gastric crises.

TREATMENT. Between June 8, 1922, and October 29, 1924, Swift-Ellis method was used, thirty-six administrations being given. At the end of the treatment the ataxia of the patient had improved, and he was able to walk with a cane. His lightning pains were less severe; his gastric crises were absent; and his general condition was better. He still had incontinence of urine and feces, and impotence; was still easily fatigued; Romberg's sign was still

SUMMARY OF THE RESULTS OF TREATMENT—CASE 6

Signs and Symptoms	Before Swift-Ellis Treatment	After Swift-Ellis Treatment	After Electrical Treatment
Romberg's sign	Marked	Marked	Absent
Knee jerk	Absent	Absent	"
Ankle jerk	"	"	"
Finger-to-nose test	Inaccurate	Inaccurate	Accurate
Heel-to-patella test	Unsteady and inaccurate	Unsteady and inaccurate	"
Pupils	Unreactive to light, sluggish to distance	Unchanged	Unchanged
Gastric crises	Severe	Absent	Absent
Explosive vomiting	"	"	"
Lightning pains	"	Less severe	"
Fatigue on exertion	Marked	Less marked	Normal
Gait	Markedly ataxic	Improved	"
Sexual power	Absent	Unchanged	"
Bladder	Uncontrolled	"	"
General condition	Poor	Better	Excellent
Weight	120 lbs.	120 lbs.	170 lbs.

GENERAL SUMMARY OF CASES

Signs and Symptoms	After Swift-Ellis Treatment	After Electrical Treatment
Romberg's sign	All cases, marked	All cases, absent
Knee jerk	All cases, absent	Cases 1, 2, 3, present
Ankle jerk	All cases, absent	Case 3, right, present
Finger-to-nose test	All cases, unsteady and inaccurate	All cases, steady and accurate
Heel-to-patella test	All cases, unsteady and inaccurate	All cases, steady and accurate
Pupils	All cases, unreactive to light, reactive to distance	All cases, unchanged
Gastric crises	Cases 1, 4, severe; cases 2-6, absent	All cases, absent
Clitoral crises	Case 1, present	Absent
Lightning pains	All cases, severe	All cases, absent
Fatigue on exertion	All cases, severe	All cases, normal
Sensory disturbances	Cases 1, 3, 4, 5, marked	Improved
Headaches	Cases 1, 2, 3, 4, severe	Absent
Morning nausea and vertigo	Case 5, for twenty years	"
Gait	All cases, markedly ataxic	Normal
Sexual power	Cases 3, 4, 5, 6, absent	"
Incontinence of urine	Cases 1, 2, 3, 5, 6, present	Absent
Incontinence of feces	Case 6, present	"
General condition	All cases, poor	All cases, excellent
Weight	All cases, much reduced	All cases, normal

marked; the knee jerk and ankle jerk were absent.

On January 14, 1925, electrical treatment was begun and continued until June 30, 1926, making three applications a week, as described in Case 1. At the end of the treatment the patient was practically normal.

The patient was seen on April 23, 1927. He had been working steadily since the cessation of the electrical treatment, and had maintained all his gains. His condition was excellent. The spinal-fluid Wassermann reaction was negative; the blood Kahn reaction negative. The cell count was 3. The colloidal-gold reaction was 000000000.

This record shows that my experimentation in the treatment of the advanced sero-negative tabetic began in the autumn of 1921. Five cases were treated in the years 1921-1923. A sixth case, treated in 1925-1926, is included because of the unusual symptoms. The results of experimentation with the five cases were so satisfactory that in the spring of 1922 I began to treat all my serologically negative tabetics electrically, and have continued to do so with the same success. At the end of the treatment these patients have all been able to resume their previous occupations, or in some satisfactory manner to be self-supporting.

Discussion of the mechanism by which these results have been produced is apart from the purpose of this paper. It is simply desired to record the unmistakable rehabilitation of these six advanced sero-negative tabetics by the use of electricity as herein described.

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LATERAL ROENTGENOGRAMS OF THE PELVIS AND THE MENSURATION OF THE CONJUGATA VERA*

BY HERBERT THOMS, M.D., F.A.C.S.†

THE determination of the diameters of the superior strait by means of the x-ray and the study of lateral roentgenograms of the pelvis have been for some time established procedures in this clinic. The results of these manoeuvers have been such that at the present time we do not feel that a contracted pelvis or a suspected contracted pelvis has been adequately surveyed unless the following measures have been carried out:

First, the determination of the usual pelvic measurements, that is, the interspinous, intercristal, intertrochanteric, external conjugate and diagonal conjugate diameters; second, the mensuration of the pelvic outlet after the manner described by me in 1915[1] and fully delineated in the present edition of Williams' *Textbook of Obstetrics;* third, the determination of the pelvic diameters of the superior strait according to the technic first described in a communication in 1922[2] and latterly in 1927[3]; fourth, the viewing of a lateral roentgenogram of the pelvis in accordance with the method described in 1927[4], and here elaborated. With these procedures, we feel that greater knowledge of the bony pelvis in the living subject is obtained than has hitherto been possible.

It is obvious to the writer that in certain instances such extensive study may not be necessary. The x-ray examination may therefore be simplified in cases where a less complete survey is deemed adequate. It is the purpose of this paper to stress particularly the value of lateral roentgenograms of the pelvis and to point out that in addition to the advantages mentioned in the previous communication, mensuration of the conjugata vera is also possible.

The following advantages may, therefore, be claimed for the procedure herein described;

1. The conjugata vera may be measured with accuracy.

2. The size and contour of the anterior surface of the sacrum may be viewed (important in the recognition of sacral abnormalities, especially those due to rhachitis).

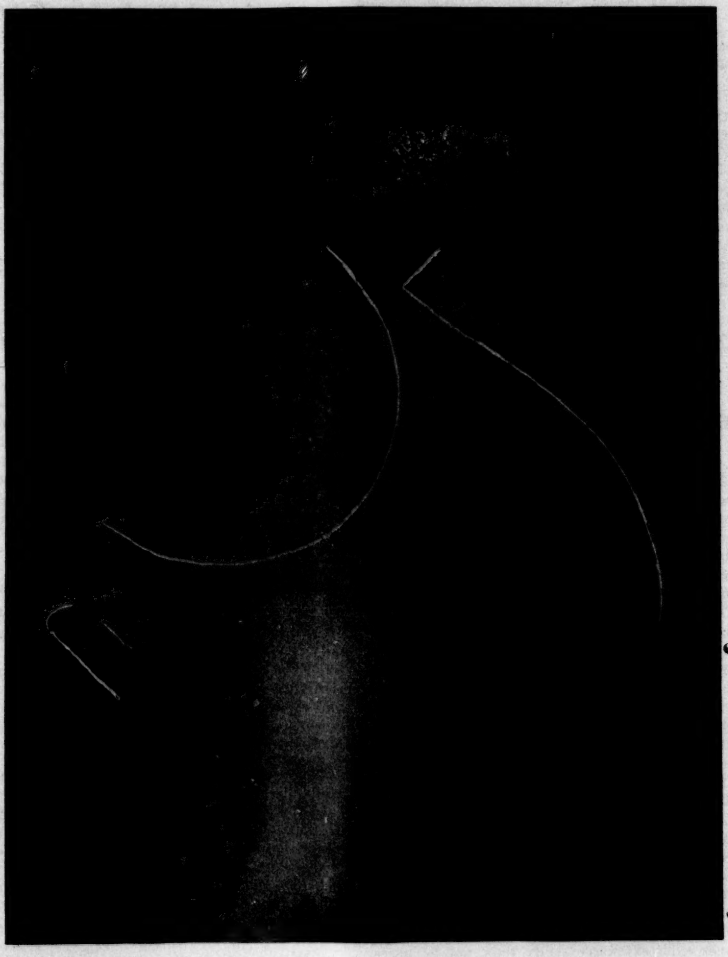
3. The relation of the presenting part to the birth canal may be studied in patients at term (an especially valuable procedure in cases where non-descent of the fetal head is present and disproportion suspected).

The technic of securing lateral roentgenograms of the pelvis is not complicated. The patient is placed laterally in the position used when the external conjugate diameter is measured. It is important that one acetabulum accurately *superimpose* the other, and this may be accomplished by observing that the legs are perfectly straight and that the nates are in the same vertical plane. By means of a pelvimeter in position for measuring the external conjugate, that is, with one end on the superior border of the symphysis and the other in the depression under the fifth lumbar vertebra, a mid-point is reached between the ends of the pelvimeter. The tube or target is centered by means of a plumb line over this point. This distance is 30 inches from the plate. The amount of cur-

*From the Woman's Clinic, Yale University School of Medicine.

†For record and address of author see "This Week's Issue," page 839.

rent is 20 milliamperes and the exposure varies from 20 to 40 seconds, depending upon the size of the patient. Duplitized superspeed films, double screen with Bucky diaphragm, and a medium focus Coolidge tube are used.



Lateral Roentgenogram at Term. Fetal head at brim, no bony disproportion. The central line of dots represent "Corrected centimeters" (see text).

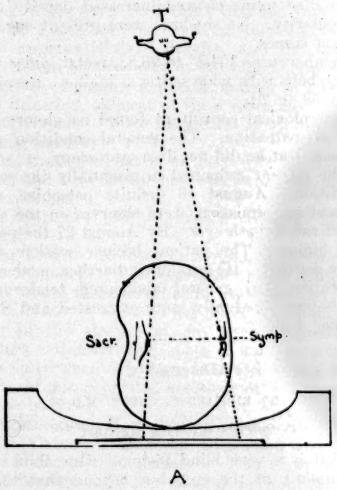
Following the exposure, the patient is removed from her position, the plate and tube being left in place. A perforated lead sheet, with small perforations 1 cm. apart (shown at B Fig. 1), is placed in the same plane as that of the antero-posterior diameter of the superior strait, and a flash exposure made on the same plate.

On development of the plate or film, the promontory and anterior surface of the sacrum and the posterior surface of the symphysis are viewed. Through the center of the plate will be seen a somewhat widely spaced dotted line. The space between each of these dots represents 1 cm. on the plane through which passes the

antero-posterior diameter of the superior strait. With a pair of calipers, the conjugata vera is readily determined.

It will be observed that the above technic is but the adaptation of my method of superior strait pelvimetry to that of lateral pelvic roentgenograms, and at this point I wish to emphasize that this latter method has in no way displaced the use of the former. In the superior strait pictures, not only the conjugata vera is measured but also the other diameters, including the important transverse diameter, the knowledge of which in certain instances is as important if not more so than that of the conjugata vera. To those interested in this technic, I would refer to the latest communication on that subject[4].

The principle involved in the present communication—that of measuring the conjugata vera by means of lateral pictures—is best understood by studying the accompanying sketch. A shows the position of the patient on the Bucky



A

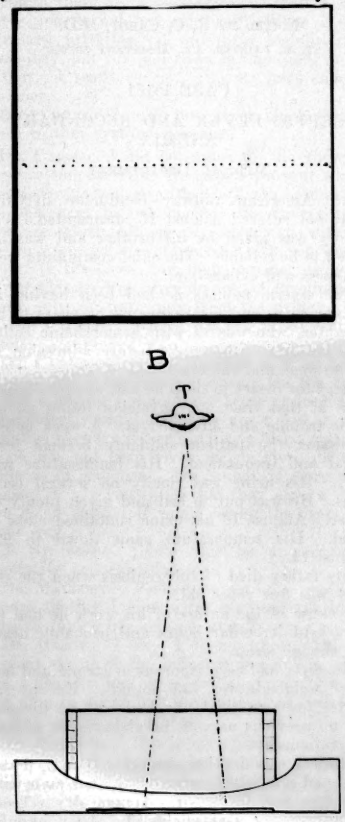
diaphragm; T, the target or tube; B, the perforated lead sheet, perforations exactly 1 cm. apart(this is made of ⅛ in sheet lead mounted on a board for rigidity). C shows the lead sheet placed over the exposed plate in the same plane as the conjugata vera of the patient. (This distance above the plate has, of course, been determined while the patient was in position before the exposure).

It is obvious that in roentgenograms made at term, not only may the relative size of the fetal head in vertex presentations be made, but in those cases where the long axis of the head lies just above or at the transverse axis of the superior strait (the R. O. T. or L. O. T. position), that a fairly accurate measurement of the biparietal diameter is possible.

SUMMARY

To repeat the principal advantages of the above method:

1. The conjugata vera in the living subject may be measured with reasonable accuracy.



C

2. The size and contour of the anterior surface of the sacrum may be viewed.

3. The relation of the presenting part to the bony birth canal may be studied in patients at or near term.

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Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 15161

SEPTIC FEVER AND SECONDARY ANEMIA

MEDICAL DEPARTMENT

An American railway conductor fifty-five years old entered August 16, disoriented. The history was given by his brother and was believed to be reliable. The chief complaints were weakness and exhaustion.

For several months he had been having irregular sleep and meals because of visits to his daughter, who was ill with hemorrhagic colitis at a hospital. Six weeks before admission he felt so weak and run down that he stopped work. Except for desire to sleep he had no other symptoms at that time. A physician found no organic trouble and advised rest. A week before admission the patient suddenly became irrational and incontinent. His temperature was 104°. His urine was cloudy on several occasions. He was put to bed and given plenty of water. August 12 his urine contained clots of blood. His temperature came down to 99° August 14.

His father died of tuberculosis when the patient was five years old.

Because of the nature of his work he had always kept irregular hours and probably never had enough sleep.

He never had been vigorous or strong and had never weighed over 135 pounds. Except for scarlet fever at thirty he had had no diseases and no previous nervous breakdowns or attacks of irrationality.

Clinical examination showed a poorly developed and nourished, pale, disoriented man with blue eyes and gray hair. Tongue dry. Teeth carious. Spine somewhat stiff. Apex impulse of the heart seen and felt in the fifth space 8 centimeters from midsternum, 1 centimeter outside the midclavicular line. Left border of dullness 9 centimeters. Sounds and action normal. A loud apical systolic murmur, heard also behind. Blood pressure 120/70 to 108/70. A few râles at the lung bases. Abdomen, genitals, extremities, pupils and knee and ankle-jerks normal. Bilateral clonus. All joints and muscles spastic, but voluntarily.

Amount of urine 25 to 38 ounces, specific gravity 1.014 to 1.010, a slight to a large trace of albumin at all of six examinations, sediment loaded with leukocytes at all examinations, occasional to many red cells at four, loaded with granular and cellular casts at two, occasional to many granular casts at four others, occasional epithelial cells twice. Two urine cultures showed colon bacillus. Renal function 5 per cent. Blood: 22,200 to 41,300 leukocytes, 92 to 91 per cent polymorphonuclears, hemoglobin 45 to 37 per cent, reds 2,575,000 to 1,580,000, moderate achromia, anisocytosis and poikilocytosis in two smears, no macrocytes, tailed forms or parasites, platelets normal once, slightly decreased once. Hinton negative. Non-protein nitrogen 59 to 68 milligrams. Widal negative. Stools negative at four examinations. Stool culture negative for typhoid and paratyphoid A and B.

By X-ray examination the kidney outlines were not visible. There were several dense shadows along the ischial spines on both sides, probably phleboliths. The outlines of both sacro-iliac joints showed increased density and irregularity. No shadows were present suggestive of stones.

Temperature 104.8° to 96.8°, rectal, pulse 146 to 80, both with wide septic swinging. Respirations 38 to 20.

A urological consultant found no abnormalities on palpation. The general condition was so poor that he did not do a cystoscopy.

The patient remained in essentially the same condition. August 18 definite petechiae, not present on admission, were observed in the conjunctivae of both eyes. By August 27 the pulse was higher. The patient became weaker and more delirous. He developed diarrhea, moderate distention and general abdominal tenderness. He became gradually more exhausted and September 4 died.

DISCUSSION

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

That is a very blind history. One thinks of pneumonia at the end, but beyond that there is no clue.

When tuberculosis is mentioned in the family history one should know whether the patient was exposed.

NOTES ON THE PHYSICAL EXAMINATION

It is very hard to tell in a man of his age whether the spine is stiff or whether it is the stiffness of age.

On the face of this record it is secondary anemia, not primary.

The non-protein nitrogen is high, but not so high as I expected.

The X-ray report states that there were no shadows suggestive of stone. This is noted be-

cause he had blood in his urine. They are thinking of the possibility of a stone in the kidney.

DIFFERENTIAL DIAGNOSIS

We have to say he has had something going on for a long time. Then something else came at the end, presumably a terminal infection. I am unable to say where that terminal infection is. The fact that he has so many leukocytes in his urine makes us wonder if it is not urinary infection. He had two colon bacillus cultures too. I cannot make any better guess than urinary infection, without saying more exactly where it is. We have nothing in the kidneys and no bladder symptoms, so that I do not see that we can be any more definite than that. Infection does not account for all that anemia. What would? I do not know what I can say except chronic nephritis considered on the basis of the renal function of 5 and the fixed specific gravity. Although the urine was not examined by the two-hour test we examined it frequently and found very little swing of gravity. So I think that the very low function, with the presence of albumin and casts, with apparently fixed gravity, and the rather high retention products in the blood, give us a diagnosis of chronic nephritis, ending in a terminal infection, as most of these cases do.

A STUDENT: Were his mental symptoms of a uremic nature?

DR. CABOT: That is an easy way to explain them, and I do not see how otherwise they could be explained.

A STUDENT: Do you expect pathology in the heart?

DR. CABOT: Nothing other than hypertrophy.

A STUDENT: With petechiae in the conjunctivae would there be an acute endocarditis?

DR. CABOT: No, I do not think we should say that in the absence of any other supporting factors. Hemorrhagic spots in anemic people, especially when they have been sick a long time with chronic nephritis, are common in any part of the body.

A STUDENT: How about general peritonitis?

DR. CABOT: "Moderate distention and general abdominal tenderness." Don't you think that is very mild for general peritonitis? It seems to me that is an understatement, if it is peritonitis.

A STUDENT: A positive culture with swinging temperature will work in with an acute endocarditis. If you could get that it would explain the whole picture.

DR. CABOT: That is a good suggestion. We should certainly say acute endocarditis if we had some other organism in culture than colon bacillus. I have never known an acute endocarditis associated with colon bacillus. It is not the right organism. Do you get that, Dr. Mallory?

DR. TRACY B MALLORY: I think I have seen it once or twice in patients already suffering from some chronic disease, a chronic type of leukemia for instance.

DR. CABOT: Yes, I think this patient has a chronic disease. It might fit.

A STUDENT: Why not a severe pyonephrosis?

DR. CABOT: In pyonephrosis you expect to feel something in the region of the kidney, although there are some degrees of that disease where you do not feel anything. When I said "urinary infection" I meant to include that possibility, but I do not mean to tie myself down to that. I really do not think we have enough evidence.

A STUDENT: You do not attach much significance to that systolic murmur?

DR. CABOT: I did not finish up the question of acute endocarditis. We have a murmur which can perfectly well go with it. There is blood at the periphery in two places. We have a temperature which can go perfectly well with it. We have not got the organism which you would expect in the blood, but that does not disprove it in the least. You often do not get streptococci. Those cultures were only from the urine?

MISS PAINTER: There was one blood culture taken.

DR. CABOT: We have to argue as if we knew nothing about the blood culture. On the basis of not knowing I should say acute endocarditis was perfectly possible, but we never feel sure of it unless we have either a positive blood culture, which has not been given us, or more definite evidence of embolism. We cannot do more than suspect it. If you think that they have a positive blood culture it would swing the facts the other way.

A STUDENT: Could this be a malignant vegetative endocarditis?

DR. MALLORY: Perhaps to get on a perfectly square footing I had better say that the blood culture during life showed an influenza bacillus.

DR. CABOT: I see. I do not know what the present status of the influenza bacillus is. The last I heard it had no status whatever. No one was able to say it was the cause of any disease. Is that the present status?

DR. MALLORY: Not in my opinion.

DR. CABOT: At any rate it is not a bacillus that has often been reported with acute endocarditis. It has been reported, especially by Libman. It is rather a rarity. But with this fact known I should say on the whole that there was a vegetative endocarditis, although I do not feel entirely positive of it. Thence we might suppose extensive infarcts of the kidneys such as to reduce this kidney function to 5 per cent, without nephritis. We could account for the anemia on the ground that this process has been going on for a long time, a septic type of anemia, which in chronic vegetative endocarditis, chronic bacterial endocarditis, we often get. We then have to account for his cerebral symptoms as part of his general sepsis or by supposing small

local lesions there. It seems to me the diagnosis lies now between two things. Either it is a chronic nephritis with a terminal infection of some kind, or it is a chronic bacterial endocarditis with infarcts destroying the function of the kidney, or it is both diseases at once. On the whole I am inclined towards the chronic nephritis, but that does not exclude the possibility of endocarditis as a terminal factor. (1) You can say he had acute endocarditis with its consequences and nothing else. (2) You can say he had chronic nephritis with a terminal endocarditis or (3) merely nephritis with some other terminal infection. I think on the whole I incline to the last. I am inclined to think that he did not have an acute endocarditis, but I do not think any human being can be at all sure. I think he did have chronic nephritis together with either infection from below or infarcts from above. Urinary infection it seems to me natural to assume in any case.

A STUDENT: If it was from infarct would you expect to get casts and albumin in the urine?

DR. CABOT: Yes. You would also expect to get blood with infarct, if the infarct were big enough to destroy the function of the kidney. On the whole I am for nephritis and against infarct.

A STUDENT: Is it very often that a kidney will be diseased enough to produce death without producing uremia beforehand?

DR. CABOT: I do not believe I can answer that,—that is, whether with a bilaterally diseased kidney a nonuremic death can occur or whether it is always a uremic death.

A STUDENT: Do you expect two independent diseases in a case like this?

DR. CABOT: No. He has a chronic disease which does something to the body whereby the usual processes of immunity are weakened, and then you get the diseases that are always ready to go across the frontier.

A STUDENT: Could that be a lymphatic involvement through the kidney from the bowels? They found the colon bacillus.

DR. CABOT: I do not see any reason to object to that.

A STUDENT: Don't you expect a higher blood pressure here,—that is with chronic nephritis?

DR. CABOT: Not with so much anemia as he now has and with as much infection as I assume he has. Those two things might well bring the blood pressure down.

A STUDENT: Would you not expect the nonprotein nitrogen to be higher?

DR. CABOT: That is one of the biggest holes to be punched in my diagnosis. I do not know why his nitrogen is not higher. But if I try to suppose anything else that would knock the renal function down it would also tend to put the nitrogen up.

A STUDENT: How long before death was the blood culture taken?

MISS PAINTER: It was taken August 20.

DR. CABOT: He died in two weeks.

BACTERIOLOGIC REPORT AUGUST 20

Blood culture shows a small Gram-negative bacillus similar to the bacillus influenzae or the bacterium melitensis.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Pyelonephritis.
Septicemia.

DR. RICHARD C. CABOT'S DIAGNOSIS

Chronic nephritis.
Terminal infection.

ANATOMIC DIAGNOSES

1. *Primary disease.*

Acute endocarditis, probably bacillus influenzae.
Acute myocarditis.

2. *Secondary or terminal lesions.*

Bronchopneumonia.
Focal glomerulonephritis.
Septic infarcts, kidneys and spleen.

DR. MALLORY: He did show a bacterial endocarditis which at autopsy, as during life, showed a blood culture of what apparently is influenza bacillus. There is of course a considerable group of closely similar organisms, but it fulfills all the criteria and I think it probably is a true Pfeiffer's bacillus.

DR. CABOT: What do you think is the relation between influenza bacillus and influenza?

DR. MALLORY: I am inclined to believe that it causes influenza, but I admit that this point of view is very far from being proved. I think it unquestionably is proved that it is a distinctly pathogenic organism that can cause a considerable number of different lesions such as bronchopneumonia, meningitis, occasionally endocarditis. We had one case of cholecystitis during the past year that gave a positive culture of influenza bacillus. It can be a secondary invading organism just as well as the pneumococcus and the streptococcus, quite aside from whatever it may have to do with the primary disease of influenza.

The organism also shows up in the heart valves in sections and resembles those which would grow in culture. He had infarcts in the kidney and in the spleen. The infarct in the spleen had become septic, and a general peritonitis had developed from perforation of that abscess. The kidney showed a glomerulonephritis, as well as the infarcts. It was however an acute nephritis, not chronic. There was a slight degree of meningitis, quite diffuse in character, over the meninges of both brain and spinal cord, and also small areas of softening due to septic emboli.

CASE 15162*

AN ABDOMINAL TUMOR FOLLOWING INJURY

MEDICAL AND ROENTGENOLOGICAL DEPARTMENTS

An Irish-American blacksmith sixty-one years old entered March 5 complaining of dull non-radiating pain on the left side of the abdomen following a blow under the ribs from an iron bar leaving a black and blue area for several weeks. The pain passed off; but soon the same pain returned at intervals. For the past three months it had been especially severe. He described it as "deep inside, slow, grinding." It occurred in paroxysms lasting an hour and a half to two hours, with intervals of complete remission. It was worse after meals. With it he had nausea at times. He had increasing eructations, marked loss of appetite, and during the past two weeks clay-colored stools. In the past eight months he had lost thirty-five pounds, with a corresponding loss of strength. Since the onset he had passed urine six or more times at night.

At a visit to the Out-Patient Department February 20, two weeks before admission to the wards, X-ray examination with a barium meal showed the stomach to be somewhat high, normal in outline, freely movable, with no filling defects. There was a pressure defect along the median portion and a palpable mass well below this area. The duodenal cap was normal. At a six-hour examination the stomach was empty. The head of the meal was in the cecum. The mass in the left side of the abdomen apparently did not arise from the stomach. Examination of the colon was recommended.

Clinical examination in the wards March 5 showed very little except a hard, tender, freely movable mass the size of a grapefruit in the left upper quadrant. The entire left side of the abdomen was tender. The Wassermann was negative. Repeated examinations of the stools showed them to be clay-colored. His temperature was irregular, varying from normal to 102°. The leukocyte count was 18,500 and the red count 4,170,000.

X-ray examination with a barium enema was done. The barium passed readily to the junction of the transverse colon with the splenic flexure. At this point there was an irregular constriction which at no time filled out. The colon at this point was in apposition with the palpable tumor mass and moved with the mass. The barium did not pass beyond the middle of the transverse colon, probably because a sufficient quantity could not pass through the constricted area. The findings are suggestive of a lesion in the distal portion of the transverse colon, although they could be produced by adhesions between the colon and the palpable mass.

*Discussed at the Roentgenological Seminar March 27, 1929.

Operation was done. Following it the patient had a moderate amount of abdominal distention and slight drainage with a stercoraceous odor, accompanied by hiccup and rise in temperature. The pulse remained strong and full. The fluid intake was small. He was given salt solution and glucose. He never rallied, and died six days after the operation.

DISCUSSION

BY GEORGE W. HOLMES, M.D.

The deformity in the transverse colon seen in the films is rather characteristic. The point in question is whether it is due to pressure from the outside or to something within the gut. That question cannot always be answered. The amount of dilatation and the character of the deformity may be as suggestive of one as of the other. Lesions inside the gut usually cause more obstruction and less deformity than those outside. The size of the mass need not influence us in deciding whether it is inside or outside. Metastases in the regional glands may be larger than the primary tumor. I should depend more upon the character and degree of obstruction than on the size of the mass.

DR. LESLIE K. SYCAMORE: If the tumor is inside the bowel should you expect more obstruction from it? The dilatation is only questionable.

DR. HOLMES: Cancer inside the gut is more likely to obstruct than outside.

DR. CLARENCE MCPEAK: How do you explain the absence of dilatation?

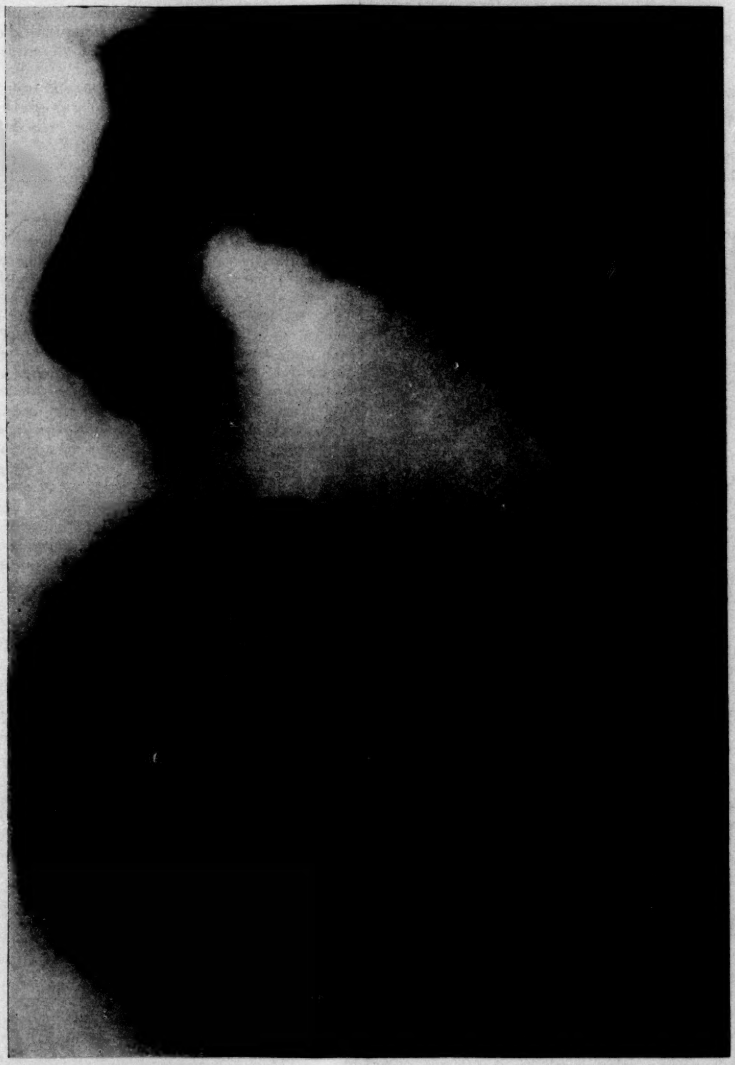
DR. HOLMES: I do not. Perhaps we should consider this in favor of a lesion outside the bowel. On the other hand the character of the deformity is more like a lesion within the bowel.

DR. SYCAMORE: He was operated upon. An inoperable condition was found—a large mass involving the transverse colon. An ileosigmoid anastomosis was done. This failed, however, to relieve his symptoms. He went downhill rapidly and died.

DR. A. O. HAMPTON: He did not have a positive guaiac?

DR. SYCAMORE: No. Several examinations of the stools were made. All were negative.

DR. HOLMES: This man had a lesion apparently arising in the large bowel which later was found to have invaded the small bowel and the wall of the stomach, the whole forming a great mass of cancer tissue. Two points of interest are (1) the history of severe trauma followed by the appearance of a tumor at the site of the injury, and (2) the absence of blood in the stools, both of which would lead away from the diagnosis of malignant disease. The X-rays however are fairly conclusive. From the point of view of the radiologist there are also two points of interest. First, we failed to show whether the mass was entirely outside the stom-



CASE 15162. Barium enema. At the junction of the transverse colon with the splenic flexure there is an irregular constriction which at no time filled out. The colon at this point is in apposition with the palpable tumor mass and moved with the mass. The barium did not pass beyond the middle of the transverse colon, probably because a sufficient quantity could not pass through the constricted area.

ach or not. There is no evidence in the notes that it was adherent to the wall of the stomach. Whether or not we ought to have showed that it was I am not sure. The mass should have interfered with the peristaltic wave as it moved over the stomach. Secondly, we failed to find the communication with the small bowel and to show that the small bowel was involved in the tumor. The latter evidence would have been of considerable value, because the anastomosis

to the small bowel gave no relief. The small bowel was obstructed. If we had known that the mass included the small intestine it might have changed the whole situation.

DR. MCPEAK: Can there be involvement of the stomach without the mucosa being involved and with normal looking rugae? We do gastrointestinal examinations during which there is no peristalsis, but still a perfectly normal looking stomach with normal rugae.

DR. HOLMES: It is a safe rule never to let the patient off the table until you have seen a peristaltic wave pass over the stomach.

DR. JOHN SPROULL: I wonder if we ought not to have had a twenty-four hour observation. Would it have helped or not?

DR. HOLMES: That is a good point. I think we are a little at fault in not making this examination more often. Whether it would have helped or not in this case I do not know; but it might have shown obstruction in the small bowel.

DR. HAMPTON: I do not see that they have proved in any way that he had obstruction.

DR. SYCAMORE: There was distention.

DR. HAMPTON: You do not demonstrate that in the colon film.

DR. RICHARD DRESSER: Where was the head of the barium column at the six hour examination?

DR. SYCAMORE: In the cecum.

DR. HAMPTON: Was there a pre-operative diagnosis of splenic hemorrhage?

DR. SYCAMORE: No.

DR. HAMPTON: The operating surgeon was not sure that the mass was not inflammatory. There was no blood in the stool and no dilatation of the colon demonstrated.

DR. SPROULL: With the twenty-four hour examination you could have found dilatation or stasis on the proximal side of the growth.

DR. HAMPTON: When can you say that there is stasis?

DR. HOLMES: That is a difficult question to answer. There are many things to consider besides the position of the barium column. I should like to see the plate taken six hours after the meal. I know that I am not very careful about checking the head of the barium column, and I should not put much faith in the statement made here.

DR. SPROULL: You do not see much in the film except the deformity. I believe that at the end of twenty-four hours you would have found stasis.

DR. HOLMES: It is important to have all available data. An enema is given and a constriction is found. A barium meal is given and at the end of twenty-four hours the head of the barium column has not passed the constriction. This is pretty good evidence of obstruction. You are much more likely to show the deformity with the enema than with the meal. The natural course of food through the bowel is less likely to be blocked than the unnatural course taken by the enema. There was obstruction to the passage of the enema in this case. We have no evidence of obstruction to the passage of the meal.

DR. SPROULL: If the mass obstructed the small bowel as well as the colon, the dilatation should be above the constriction in the small bowel.

DR. HOLMES: Yes; and it is possible that an observation made twenty-four hours after giving the barium meal would have demonstrated it.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Carcinoma of the transverse colon.
Peritonitis.

ANATOMIC DIAGNOSES

1. *Primary disease.*

   Carcinoma of the transverse colon with metastases to the lung.

2. *Secondary or terminal lesions.*

   General peritonitis.
   Thrombosis of the right iliac vein.
   Operative wound—anastomosis of the ileum to the sigmoid.

DR. TRACY B. MALLORY: The post-mortem examination showed a large annular carcinoma of the transverse colon with raised firm edges and an ulcerated center. The tumor had invaded the gastrocolic omentum and was firmly adherent to the exterior of the stomach. It had not invaded the stomach wall, however. Adhesions had formed between the tumor and two coils of small intestine, and the tumor had invaded these loops and caused fistulae between them and the lumen of the colon. The surgical anastomosis between the ileum and sigmoid was intact. A slight general peritonitis was present, which did not however seem to have any connection with the operative wound. The liver was large but free from metastases, and there was no obstruction of the bile ducts. The gall bladder contained thick black bile. No explanation for the clay colored stools was found. A small metastatic tumor nodule one centimeter in diameter was found along the anterior edge of the right lower lobe of the lung.

A terminal complication was a thrombosis of the right iliac vein with marked swelling of the right leg.

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TRAUMA IN MALIGNANT DISEASE

ONE of the most disputed points with regard to the etiology of malignancy is the rôle played by a single injury to a part in which a tumor subsequently develops. The medico-legal importance of this possible factor is very great, due to cases arising in connection with workmen's compensation and with automobile and other injuries. The question has been almost hopelessly clouded by uncritical reports of thousands of cases of tumor supposedly due to trauma.

Almost the first thorough and unprejudiced study of this subject was that of Thiem[1], who laid down certain postulates that should be fulfilled to establish trauma as an etiological factor. These were that the occurrence of the accident must be proved, that the injury must have been sufficiently severe to affect the tissues, that the tumor must be definitely related to the site of injury, and that the tumor should not appear until a reasonable time after the occurrence of the trauma. When these criteria are applied but few of the cases in the literature are valid. Ewing[2] has further refined these requirements by demanding that the previous integrity of the wounded part be shown and that the tumor be of a type that could result from trauma. The general usage at present appears to be to recognize sarcomata as possibly associated with single traumas but to eliminate the group of epithelial tumors.

One of the most important factors to be considered from the clinical angle is that most tumors are painless until they have reached a considerable size and very probably in almost all the cases any trauma to the part merely called attention to a pre-existing tumor rather than causing its development. It has been pointed out by a number of German pathologists that in spite of the tremendous number of war injuries there were no more tumors among the war veterans than in civilians.

Knox,[3] in a valuable review of the material on this subject, comes to the conclusion that the real damage done by a single injury is the stimulation of a pre-existing tumor to greater activity of growth or the determination of site of metastases of an already existing tumor.

1 Thiem: Handbuch der Unfallerkrankungen. Stuttgart, 1909.
2 Ewing, J.: Am. J. Surg., 40:30, 1926.
3 Knox, L. C.: Arch. Path., 7:274, 1929.

EPIDEMIC SEPTIC SORE THROAT

THE clinical, etiological, epidemiological, and pathological studies which have been concentrated on septic sore throat have placed this disease among those of which medicine can claim to have cleared away uncertainties as to its causes, behavior and prevention. As shown by White* in the first article in this number much attention has been devoted to this disease since 1875. He records over sixty epidemics in this and other countries with definite records and estimates amounting to nearly thirty thousand cases. To the importance of great numbers of sufferers the mortality rate adds much to the burden imposed on individuals and communities. It is a disease which kills. To this might be added a considerable number of epidemics with no record or estimate of the morbidity and mortality and Drs. Holmes† and Bigelow and White‡ present a record of our recent spectacular outbreak in this state.

Modern science has demonstrated that the disease is due to the streptococci, usually from an infected udder and sometimes undoubtedly from an infected milker who may directly plant the germ in the milk or convey the disease to the udder when it continues to flourish.

The disease is often malignant, causes severe suffering and a considerable mortality depending on the virulence of the streptococcus and the resistance of the host. Fortunately it is preventable because efficient pasteurization kills the

*Page 797.
†Page 805.
‡Page 807

streptococcus, and the effect of its presence in milk can often be recognized by careful inspection of the strainer through which the milk has passed. When detected, the diseased carrier, whether cow or herdsman, can be dealt with to prevent further extension of the disease if expert advice is sought. Unfortunately the producer may not believe in pasteurized milk for three reasons: (a) it costs more, (b) it involves some added work and (c) some of his customers want unpasteurized milk. Hence if he is not imbued with a sense of responsibility or does not know of the danger to consumers the disaster may not be avoided or checked until the explosion has occurred and enough persons are found to be ill to call for investigation by public health officials.

We have repeatedly advocated the use of pasteurized milk but there is not by any means universal endorsement of this contention. We shall probably continue to have outbreaks of septic sore throat and other diseases due to the vehicular function of this potent culture medium until consumers are convinced that there may be a serious danger incident to drinking raw milk.

Some members of the legislature and a few doctors are still unconvinced. Iteration and reiteration must be the order of the day until more general conversion has been brought about.

People still have the privilege of themselves using unsafe milk but worse than that of giving it to children.

TRANSPORTATION TO THE MEETING OF THE AMERICAN MEDICAL ASSOCIATION

In less than three months the American Medical Association meets in Portland, Oregon. The trip is long but is one offering many attractions. It may be made a particularly pleasant trip if the company is congenial. With these facts in mind the Delegates from New England have been consulted and look with favor on a plan to have special cars in which to go and return.

The Northern Pacific Railroad is anxious to supply this service. On the journey west there would be granted the option of a four day trip through Yellowstone Park. The return trip would be by way of Seattle, Puget Sound, Vancouver and the Canadian Rockies, with stops at the cities mentioned and at Lake Louise.

The railroad fares are low, much less than the fare and a half rate commonly paid for attendance at conventions. Service and meals are far superior to those supplied by the railroads of New England, or in general by railroads east of Chicago.

The JOURNAL hopes that enough members of the New England State Medical Societies may desire to attend the meeting to fill several cars even if not a special train. In any event the cars would probably be part of a special train, this giving a chance to meet physicians from other parts of the country.

Full details of the trip as outlined will be published in the JOURNAL. All who are interested are urged to write promptly to the Headquarters of the Massachusetts Medical Society, 126 Massachusetts Avenue, Boston. A letter or posteard will imply no obligation on your part but will help greatly in making plans if New England physicians wish to be together and be relieved of the details of travel.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

WHITE, BENJAMIN. Ph.B., Ph.D. Yale. Director, Division of Biologic Laboratories, Department of Public Health, Commonwealth of Massachusetts. Assistant Professor of Bacteriology, Immunology and Preventive Medicine, Harvard School of Medicine and Harvard School of Public Health. Honorary Member American Academy of Arts and Sciences. His subject is: "Epidemic Septic Sore Throat, I. Historical Review." Page 797. Address: 375 South Street, Jamaica Plain, Mass.

HOLMES, MAY SALONA. M.D. New York Infirmary for Women and Children, 1895. Superintendent and Resident Physician, Belmont Hospital, Worcester. Her subject is: "Epidemic Septic Sore Throat. II. A Clinical Study of the 1928 Epidemic in Massachusetts." Page 805. Address: Belmont Hospital, Worcester, Mass.

BIGELOW, GEORGE H. A.B. M.D. Harvard 1916. Doctor of Public Health 1921, Commissioner of Public Health Massachusetts. Address: State House, Boston. Associated with him is:

WHITE, BENJAMIN. Their subject is: "Epidemic Septic Sore Throat. III. Review of the 1928 Epidemic in Massachusetts." Page 807.

CUSHING, HARVEY. A.B., A.M., LL.D., D.Sc., M.D. Harvard Medical School 1895. F.A.C.S. F.R.C.S., England and Ireland, Surgeon-in-Chief at the Peter Bent Brigham Hospital, Member of the American Surgical Association and the American Academy of Arts and Sciences. Address: Peter Bent Brigham Hospital, Boston, Associated with him is:

MINER, LEROY M. S. D.M.D., M.D. Boston University 1907. F.A.C.S. Professor of Clinical Oral Surgery, Dean of Harvard Dental School. Associate Professor of Stomatology at Boston University. Address: 363 Marlboro Street, Boston. And

COUES, WILLIAM PEARCE. M.D. Harvard, 1894. F.A.C.S. Surgeon to Out-Patients, Massachusetts General Hospital. Consulting Surgeon, Massachusetts Eye and Ear Infirmary. Instructor in Surgery, Tufts College Medical

School. Address: 12 Monmouth Court, Brookline. And

BOCK, ARLIE V. A.B., M.D. Harvard, 1915. Associate Physician, Massachusetts General Hospital. Assistant Professor of Medicine, Harvard Medical School. Address: Massachusetts General Hospital, Boston.

LEWIS, FREDERICK T. A.B., A.M., M.D. Harvard, 1901. Associate Professor of Embryology, Harvard Medical School. Formerly, Editor, American Naturalist. Associate Editor, Anatomical Record. Address: Harvard Medical School. And

WHEELER, W. M. Ph.D., S.D., LL.D. Dean of Bussey Institution. Address: Bussey Institution, Forest Hills, Boston. They contribute a "Symposium on John Hunter." Page 810.

LUCE, LEROY A. M.D. Tufts College Medical School, 1906. Assistant Visiting Physician for Immunology, Boston City Hospital. Consultant in Neuro-Syphilis, New England Hospital for Women and Children. Formerly First Assistant Visiting Physician for Diseases of the Nervous System, Boston City Hospital and formerly Physician-in-Chief, Electro-Therapeutic Department, Boston Dispensary. His subject is: "Is The Rehabilitation of the Advanced Sero-Negative Tabetic Possible?" Page 823. Address: 416 Marlborough Street, Boston.

THOMS, HERBERT. M.D. Yale, 1910. F.A.C.S. Associate Professor of Obstetrics and Gynecology, Yale University School of Medicine. His subject is: "Lateral Roentgenograms of the Pelvis and the Mensuration of the Conjugata Vera." Address: New Haven Hospital, New Haven, Conn.

THE GRADUATE COURSE IN CANCER

THE Committee in charge of the proposed Graduate Course in Cancer to be held in Boston April 23-25th, reports a registration of 190. As we go to press, 104 of the registrants being physicians and 86 dentists, the latter registering for the first day only. The limit of capacity has been reached in several of the clinics where available space is unfortunately restricted.

Only casual reference having been made in these columns to the so-called Canti film, which will be shown on the afternoon of April 25th, our readers may be interested to know that this picture, consisting of three reels, was produced by Dr. R. G. Canti as part of an investigation of Cancer which is being carried out at the Strangeways Hospital, Cambridge, England, and at St. Bartholomew's Hospital in London.

It was thought that through the cinema it would be possible to obtain records over long periods of time which could be examined in much shorter periods and re-examined repeatedly at varying rates of speed or even studied in the reverse order of time, thereby tracing to their origin changes which might have taken place.

The film shows, at first under low power, later under a high degree of magnification the actual processes of growth and reproduction in living cells cultivated from the periosteum of the fowl embryo, and in contrast, the cell activities in tissue from a rat sarcoma. The normal cells appear in various forms, sometimes in the process of mitosis, always in what is apparently their usual state of intense activity.

Toward the end of the film the action of irradiation is brought out. Within twenty minutes after the application of radium emanation all motion among the cells has ceased. This happens alike with the normal cells and with the sarcoma cells, but in the latter the destruction appears to be more complete. This observation has been interpreted as lending support to the theory that radium has a selective action with relation to the cells of a malignant tumor.

The Committee is heartily gratified by the enthusiastic response to this endeavor to meet an expressed demand from certain members of the profession and offers its assurance that every effort will be put forth to provide a program commensurate with the time expended by those who attend the Course.

The Committee requests that any physician in Massachusetts who has failed to receive an announcement of the Course communicate with the office of the Massachusetts Medical Society, 126 Massachusetts Avenue, Boston.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY

Frederick L. Good, M.D., *Chairman* Louis E. Phaneuf, M.D., *Secretary*
Frederick J. Lynch, M.D., *Clerk*

What Causes Sacro-iliac Pain During Pregnancy?

Changes in the sacro-iliac synchondrosis during pregnancy may cause discomfort, pain or disability. When present they usually appear during third trimester and continue until after delivery.

Williams stated that since Luscka demonstrated a bursa in the sacro-iliac synchondrosis it must be regarded as a true joint and of considerable importance during pregnancy and labor. Also the late Dr. Lovett in his text book said in reference to sacro-iliac disease, that due to softening of the cartilage and relaxation of the joint late in pregnancy, the patient may complain of backache and pain, often making walking difficult or impossible.

The patient's own description of her symptoms is usually quite characteristic. She has a low backache with more or less tenderness over

the affected joint. The pain may be unilateral or bilateral, dull or sharp and usually radiates down along the sciatic nerve. It is aggrevated by walking or turning over in bed. Very rarely does this condition persist long after delivery, although it not uncommonly recurs during succeeding pregnancies. Other conditions to be thought of in diagnosis are, back strain due to the marked lordosis with which a woman stands during the last months of pregnancy and the discomfort due to foot strain.

The treatment is rest; lying flat in bed usually gives prompt relief, perhaps with an extra pillow under her lower back for added support. Strapping may be tried, also a properly fitted corset or belt sometimes gives considerable comfort. Patient should avoid much walking and should not allow herself to become over-tired.

Questions of a similar nature to the above will be discussed in the JOURNAL each week until June. They may be addressed to the Clerk of the Committee, in care of the JOURNAL and will be answered by members of the Committee of the Section of Obstetrics and Gynecology.

MISCELLANY

PRIZES AWARDED BY THE JUDGES IN THE PLAYWRITING CONTEST

The Massachusetts Tuberculosis League announces that the Judges in the Fourth Annual Health Playwriting Contest have awarded prizes as follows:

1st Prize—"Times Have Changed"—Concord High School.

2nd Prize—"A Little Child Shall Lead Them"—Melrose High School.

3rd Prize—"Temple of the Soul"—Durfee High School, Fall River.

Honorable mention was also given by the Judges to the high schools of Fairhaven, Provincetown and Lowell.

The Judges of this year's Contest were Dr. John B. Hawes, 2nd, of Boston, Chairman, Mrs. Carl L. Schrader of Belmont and Mr. Richard A. Schmoyer of the Lynn Public Schools.

A VAST ARMY OF THE DEAFENED

The actual prevalence of deafness is difficult to measure, for the sufferer, either unwilling to acknowledge his social handicap or unaware of the serious progressive nature of the affliction, often waits until his hearing is irremediably impaired before seeking medical aid. A conservative estimate of the extent of defective hearing, based on group tests of school children and drafted men, places the number of persons now suffering from impaired hearing in the United States at 10,000,000. Of these, 3,000,000 are school children with a lifetime before them; a lifetime which must be a continual struggle against bitter disappointments, misery and want. In the light of these figures, humanitarianism demands that science turn immediately to this problem and seek ways of alleviating the suffering and social maladjustments that deafness causes among its victims.

The American Otological Society, recognized in America and abroad as the authoritative senate on diseases of the ear, initiated a research movement a few years ago to study the causes of chronic progressive deafness. In 1926, the Carnegie Corporation made a grant of $90,000 to the Scientific Committee of this group for the investigation of otosclerosis, a form of chronic progressive deafness. This grant, extending over a five-year period, was awarded in amounts decreasing each year with the expectation that the Society could build up an independent fund to expand and carry on the research. To this end a campaign is under way to raise a permanent endowment fund of $2,500,000 to be devoted to this work.

If this important work is to go forward without interruption, and the effort already expended is to be conserved, a permanent fund of $500,000 must be built up immediately. This can only be done through the co-öperation of every philanthropic man and woman who has the sympathy and vision to realize the seriousness of the problem.

THREE NAVAL HOSPITALS FOUND TO BE CROWDED

Crowded conditions were found in naval hospitals in Newport, Brooklyn and Boston by the Naval affairs Sub-committee of the House Appropriations Committee during its recent inspection of naval facilities on the Atlantic Seaboard, Representative Burton L. French of Idaho, chairman, declared recently.

When the 1931 Navy Department appropriation bill is taken up by the Appropriations Committee, Mr. French intimated, an attempt will be made to increase facilities in the hospitals. Most of the crowded condition is caused by the diversion of Veterans' Bureau cases to naval hospitals.

Overcrowding in the Newport Hospital eventually will be relieved by the construction of the new Southern New England Veterans' Bureau Hospital near Hartford, but the Appropriations Committee, as a result of the inspection, will be prepared to increase the Newport facilities until the new hospital is ready to take over the additional patients, it was indicated.—*New York Times.*

GAIN IN PREVALENCE OF SPINAL MENINGITIS AND SCARLET FEVER

Further increases in the prevalence of cerebrospinal meningitis and scarlet fever were reported to the Public Health Service by State health officers in statistics on the incidence of communicable diseases for the week ended March 16.

Reports from 45 States showed 325 cases of spinal meningitis for the 1929 week as compared with 155 cases for the same week of last year. Reports from 46 States showed 5,947 cases of scarlet fever for the week of 1929 and 5,017 cases for the corresponding week of 1928.

DEFECTS OF SPEECH

It is estimated that in the United States at least 1,000,000 persons have some form of speech disorder, and of these, approximately 500,000 are school children who stammer or stutter, the Public Health Service announced April 8.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1928 AND SEVEN YEAR AVERAGE MONTH ENDING MARCH 16

DISEASES	1929					1928			
	Wk. ending Feb. 23	Wk. ending Mar. 2	Wk. ending Mar. 9	Wk. ending Mar. 16	Average cases reported for week corresponding to March 16 for past seven years.	Wk. ending Feb. 25	Wk. ending Mar. 3	Wk. ending Mar. 10	Wk. ending Mar. 17
Actinomycosis	-	-	-	-	-	-	-	-	-
Anthrax	-	-	-	-	-	-	-	-	-
Botulism	-	-	-	-	-	-	-	-	-
Cerebrospinal Men.	1	2	5	2	1	2	1	-	2
Chickenpox	58	80	85	106	75	72	97	113	108
Conjunctivitis Inf.	-	-	-	-	1	-	-	-	1
Diphtheria	16	26	18	17	38	26	20	23	19
Dysentery Amoebic	-	-	-	-	-	-	-	-	-
Dysentery Bacillary	-	-	-	-	-	-	-	1	-
Encephalitis Epid.	-	2	1	1	2	1	1	-	-
Favus	-	-	-	-	-	-	-	-	-
German Measles	96	134	172	167	12	-	1	3	3
Hookworm Infection	-	-	-	-	-	-	-	-	-
Influenza	99	5053	51	51	82	3	3	175	5
Leprosy	-	-	-	-	-	-	-	-	-
Malaria	-	-	-	-	-	-	-	-	-
Measles	373	381	484	553	381	358	358	377	398
Mumps	109	53	110	117	87	172	209	408	287
Paratyphoid Fever	-	-	-	-	-	1	1	-	-
Pneumonia (Broncho)*	42	54	40	42	58	35	50	43	43
Pneumonia (Lobar)	59	70	55	46	72	60	60	55	61
Poliomyelitis	-	-	-	-	-	-	1	-	-
Scarlet Fever	56	48	55	68	111	89	74	71	80
Septic Sore Throat	1	1	2	-	2	2	-	1	1
Smallpox	2	-	-	9	6	2	3	4	-
Tetanus	-	1	-	-	-	-	-	-	-
Trachoma	-	-	-	-	-	-	-	-	-
Trichinosis	-	-	-	-	-	-	-	-	-
Tuberculosis (Pul.)	19	30	31	31	36	45	33	27	59
Tuberculosis (O.F.)	2	3	1	3	6	15	4	2	25
Typhoid Fever	-	-	2	-	1	4	-	1	-
Typhus Fever	-	-	-	-	-	-	-	-	-
Tularemia**	-	-	-	-	-	-	-	-	-
Undulant Fever	-	-	1	-	-	-	-	-	-
Whooping Cough	29	15	30	34	69	175	133	156	113
Gonorrhoea	33	46	32	51	22	32	28	21	25
Syphilis	31	46	76	48	25	51	50	30	27

* Made reportable January 1, 1925.
** Made reportable July 1, 1928.
REMARKS: No cases of Asiatic cholera, glanders, plague, rabies in humans and yellow fever during the past seven years.

RECENT DEATH

HOSMER—A long and faithful record of service to humanity has been closed by the death at the Lowell General Hospital on April 8, 1929 of DR. CHARLES EDWARD HOSMER of Billerica, aged 91. Dr. Hosmer was the grandson of John Hosmer, who, at the age of sixteen, fought in Captain John Parker's company of Minute Men at Lexington. Born on May 25, 1837 in Bedford, Dr. Hosmer was graduated A.B. from Brown University in the class of 1861. Soon he entered the United States military service as a private in the 10th Rhode Island volunteers, serving in Virginia. Mustered out in September 1862 he was surgeon's steward and assistant surgeon in the Navy in 1864 and 1865, being honorably discharged in October of the latter year. Having entered Harvard Medical School in 1863 he took his degree in 1867 and settled in practice in Waltham, where he remained for the next four years, meanwhile joining the state medical society in 1869. For fifty-one years from 1872 to six years ago, Dr. Hosmer had practised medicine in Billerica, also serving the town in the school committee from 1881 to 1887 and as a representative in the Legislature in 1896-97. He was president of the Bennett library and president of the Billerica Historical Society and held membership in Post 185, Grand Army of the Republic, of Lowell, and in the Billerica Veterans' Association. Two sons and three daughters survive him.

CORRESPONDENCE

A CORRECTION WITH RESPECT TO HOSPITAL SPACE FOR THE CHILDREN OF THOSE IN COMFORTABLE CIRCUMSTANCES.

Harvard Medical School, Department of Pediatrics
Children's Hospital 300 Longwood Avenue
Boston, April 5, 1929.

To the Editor of the
NEW ENGLAND MEDICAL JOURNAL

In the JOURNAL of March 28th, relating to the Children's Hospital Contagious Unit, the editorial statement is made "It is brought out in Dr. Blackfan's statement—but no space available anywhere for the children of those in comfortable circumstances or the well to do." Clearly such a statement is not in accord with the facts. I am chagrined to find on investigation that the statement sent to the JOURNAL on March 16th had not been edited finally by me. The corrected statement as published in the C. H. News reads, "In Boston—there is *inadequate* hospital space for the children of those in comfortable circumstances—."

I realized that several hospitals have limited facilities of this sort and without them we should be very much worse off than we are. Among others, the Haynes Memorial Hospital, the Brookline Contagious Hospital, and certain rooms at the Boston City Hospital and the Newton Hospital are available. The very enumeration of the facilities at these Hospitals, however, emphasizes the *inadequacy* of the present situation. In the Haynes Memorial Hospital, according to Dr. Wesselhoeft, twenty-eight private rooms are available for patients of all ages. This Hospital serves officially twenty-seven different Boards of Health, and the Port Immigration Service. The Brookline Hospital has about nine private rooms, the Boston City Hospital has about twenty private rooms, and the Newton Hospital has about five private rooms. This limited number of rooms available for private patients is obviously small when one takes into consideration the total population of Boston and greater Boston as well.

The population normally served by the Children's Hospital is clearly very large. In addition, the Hospital is often faced by the problems which arise when patients from distant communities develop communicable diseases while under its care.

While regretting the error which might well lead to misunderstandings with other Hospitals, I am glad to have the opportunity for presenting the facts to the medical community.

Very truly yours,
KENNETH D. BLACKFAN.

THOUGHTS REGARDING NURSES BY A PATIENT

To the Editor

I have read with interest the various comments upon the facts regarding nursing as told by the report "Nurses, Patients and Pocketbooks". One impression forces itself upon me: referring to the attitude of nurses towards their work, there are mentioned among other things, desire for a fuller life, wish to have the menial tasks done by others, their right to fixed hours, etc. The answer to all this is the *Commercial Spirit*, the *Non-recognition* of the fact that nursing is different from office-work and other money-making employments. The stimulating motive must be *Religion*, *Self-Sacrifice*. Doctors show this, ministers almost universally live by it, College professors on small salaries, all do their work from the heart many with pay almost too meager to support a family. If a nurse dwells on her hours, her pleasures, her pay, let her take up stenography or bookkeeping but *don't let her undertake the care of the sick and suffering*. Once when I heard a trained nurse say, "Why should a young girl spend her life in that way!" referring to nursing a gracious, kindly lady afflicted with melancholia, I wanted to reply, "Why should our Saviour have given his life for us?"

I speak from the experience of over twenty years as a patient. In the variety of nurses which have come under my notice during that time, the following motives have been apparent:

1. Social position, or at least being called "Miss" and taking meals with the family in private houses instead of in the kitchen where many naturally belong.
2. Matrimony.
3. Dress and amusement, the high pay being taken advantage of to work only six months out of the year or every alternate year, spending the accumulated wages in dress and frivolity. The $45 a week which many a patient is forced to squeeze from an almost impossible income, goes to dress the flapper nurse in silk underwear and velvet dancing dresses. The small savings referred to generally means extravagance, not lack of means to save.

It is a relief to turn to the other side. There are still a few of the Florence Nightingale type, though the price is almost prohibitive. But they belong to the older nurses and are fast dying out.

A PATIENT.

April 7th, 1929.

A SPECIAL TRAIN FOR PORTLAND, OREGON

165 Broadway, N. Y.,
April 9, 1929.

Dr. W. L. Burrage, Secy.,
Massachusetts Medical Society,
Brookline, Mass.

Dear Dr. Burrage:

At suggestion of Dr. Olin West, Editor of the *American Medical Association Journal*, I am sending you two pamphlets descriptive of a "Golf Special" which we will operate from Chicago to Portland, also on return from Portland to Chicago for Doctors, their families and friends who will attend the American Medical Association Convention.

I will deeply appreciate it if you will post these in a conspicuous place where it can be seen by members of your Society.

An inquiry to Dr. J. P. DeWitt, 112 Shorb Avenue, N. W., Canton, Ohio, or to the undersigned will be promptly answered.

Yours truly,
H. H. GRAY.

P. S. Please note one deals with going trip; the other with return trip—passengers welcome for either or both.

NOTE:—Pamphlets on file at 126 Massachusetts Avenue.

AN EXPLANATION OF A PART OF PROF. HARWOOD'S SURVEY

412 Beacon Street
Boston, Mass.

April 4, 1929.

Editor, NEW ENGLAND JOURNAL OF MEDICINE:

In reviewing the recent Health Survey of Cambridge made by Prof. Harwood, of Technology, one of the points which he criticized and which seems to need explanation, might be of interest to some of your readers, for many of those readers run clinics where an immediate diagnosis is difficult.

It was noted in the Survey that the Cambridge Dispensary had 35% to 50% of their patients diagnosed as "suspicious," that is, tubercular suspects. I have made occasion to have a talk with Dr. Fleming, who has for a very long time self-sacrificingly directed and given his interest to this Dispensary, and he explains the above noted circumstance in a manner which interested me a great deal.

He feels that patients coming to his Dispensary have to be diagnosed as (1) non-tubercular, (2) tubercular, including the various forms, and (3) suspicious, and to make a positive diagnosis of tuberculosis you have to have either a positive sputum or a combination of other signs such as a positive Von Pirquet, a positive x-ray, contact with loss of weight, persistent cough, or some condition of surgical tuberculosis.

Many cases who come to him for examination have only one of these signs but he feels that if a suspicion is raised about them and he so diagnoses them, he can then follow them for months or years if necessary until he can safely discharge them from the Clinic. If they were immediately diagnosed as non-tubercular, he would have no mechanism through which his nurses could keep them under observation and see that they return for periodic examinations. This account seems to me justified and does explain many cases which a non-medical man might easily think should be diagnosed definitely one way or the other in days rather than months.

Very truly yours,
HILBERT F. DAY,
President, Cambridge Tuberculosis Association.

A NOTE ON THE STUDY OF HEART DISEASE IN LONDON, PARIS AND VIENNA; A LETTER FROM ABROAD.

February 12, 1929.

To the Editor of the NEW ENGLAND JOURNAL OF MEDICINE:

It has been suggested that certain glimpses that I have had of medical work in Europe during the past four months may be of interest and perhaps of value to some of your readers, and therefore I am sending you this letter It is perforce of limited scope dealing as it does primarily with a few cardiovascular observations, but with a view also of a little of the general medical situation in Europe. The stay of a few weeks in London, a few weeks in Paris and two months in Vienna is all too brief a time to permit any authoritative remarks, and I shall simply write a few impressions of what I have seen.

In London, internal medicine is better organized than it used to be, but, as in much of the rest of Europe, the depressing result of the World War is still evident, in contrast to the situation in the United States, where there is so much more equipment and personnel for medical work both in routine and in research. The English school of medicine continues its strong foundation and interest in physiological studies. It was delightful to see something again of Sir Thomas Lewis, with his able associates Grant, Craib and Cotton, actively carrying on their cardiovascular clinical and research work at the University College Hospital and Medical School. Their interest lies not only in studies of the peripheral circulation, which have absorbed a large store of Sir Thomas's time in the past few years, but in other circulatory problems as well, including Grant's pathological work and Craib's analysis of the electrical activity of muscle. The outpatient department and ward round teaching of the medical students by Sir Thomas and his associates, although simple and fundamental, always gives one much instruction and stimulation. It is also of interest that assisting Lewis and Grant at the University College Hospital this year are Drs. William J. Kerr of San Francisco and Louis Viko of Salt Lake City, both graduates of the Harvard Medical School, while last year Dr. T. D. Jones and the year before Drs. H. M. Marvin and J. E. Wood, Jr., all formerly at the Massachusetts General Hospital were their assistants.

At the National Heart Hospital in London formal graduate courses, each lasting a few weeks, are given several times a year. I had the opportunity of attending some of the classes of Drs. John Parkinson and Thomas Cotton. These men are very able clinicians and bid fair to hold in London and in England the position in cardiovascular consulting work once held by Sir James Mackenzie, whose teachings and influence appear still to be a dominating factor in cardiovascular practice in London. Dr. Parkinson also has a clinic at the London Hospital and Dr. Cotton assists Sir Thomas Lewis at the University College Hospital; Dr. Bedford whom I also met is a younger associate of theirs at the

National Heart Hospital. Dr. Geoffrey Bourne, particularly interested in cardiovascular work at St. Bartholomew's Hospital and Dr. Donald Hunter, working at the London Hospital, have both spent some time in recent years in America and it was a pleasure to see them again. In internal medicine, and particularly in neurology, London affords a chance for visitors to see clinical work of interest, but my own time was too limited to take particular advantage of these opportunities. I must not, however, leave word of London without mentioning the very interesting film recording the experiments of William Harvey as reproduced by Lewis and Dale for presentation at the Harvey Tercentenary Celebration in London last year, and which I had the opportunity of seeing at the Centennial Celebration of the University College Hospital Medical School in the Fall.

In Paris Vaquez is the leader in the field of heart disease. An able clinician, with a strong personality, he has made his influence felt not only in France but in many countries closely affiliated medically with France, like Roumania, Czechoslovakia and the states of South America. Cardiology owes much to the French, in the introduction of auscultation and in the establishment of percussion a hundred years ago, in physiological studies a generation or two ago, and in such pioneer work as that of Bordet in roentgenology of the heart and great vessels in recent years. Just now it is in the clinical field—in diagnosis and treatment—that the French physicians working in cardiovascular disease are particularly interested. It was stimulating to visit the French cardiovascular school, so different in some of its activities from those of the English and the German, and to learn a little of the work of the individual physicians all of whom were very cordial: Professor Vaquez at the Hôpital de la Pitié with his associates there, Drs. Bordet, Heitz, Géraudel, Donzelot and Giroux; Professeur Agrégé C. Lian at the Hôpital with his associate Blondell; Professeur Agrégé A. Clerc at the Hôpital Lariboisière, and Dr. Lutembacher with his particular interest in the reproduction of heart sounds and action and his talking movie (film parlant). One of the leaders of the French cardiac school not in Paris is Professor Gallavardin of Lyon whom I hope to visit in the spring.

Vienna continues to be an important medical center with its great wealth of pathological material and instruction in spite of the great post-war depression still evident throughout the country. Associated with the Socialistic government in Vienna has been the introduction of the Krankenkasse system which largely controls the practice of medicine there. It is a makeshift arrangement which has been for the most part unsatisfactory both for the medical profession and people—what its future is no one knows at present. The payment of health insurance by the Viennese allows their calling the Krankenkasse physicians when ill. The very small fees allowed the doctors for these consultations—a few schillings, amounting to 25 or 50 cents—are not conducive to the best medical practice; even the most conscientious doctors must work hard and fast to earn a livelihood. The University too, and the hospitals cannot afford to pay living wages to their faculty and staffs. As a result the situation would be very bad indeed for all but the older professors with their remunerative private practices were it not for the foreign, mostly American, physicians studying in Vienna. Organized under the American Medical Association of Vienna these English speaking students attend many courses arranged for them under the University faculty. During 1928 close to one thousand of these visiting doctors took hundreds of courses in all branches of medicine and surgery and thereby helped to support many of the ablest native physicians of Vienna. Thus there has been a double advantage—the Americans have had well arranged instruction, generally good, at a reasonable fee, and the Austrians have received a minimum of $5. an hour for their courses. Those attending the course divide up among themselves the expense—thus if a course has twenty members each one may pay as little as twenty-five cents an hour. Usually fewer are in the course and so the charge is somewhat more, along with the entrance fee for life membership in the American Medical Association of Vienna of $10. and a moderate charge monthly to pay expenses. The courses are given mostly in English, but some also in German and in themselves these latter courses are good instruction in medical German as well as in the particular medical or surgical subject. The courses in pathology and certain surgical specialties, particularly ophthalmology, and otology and laryngology, are apparently the most popular, although a good many of the students enroll also in medicine, obstetrics and other subjects. There are certain drawbacks about the American Medical Association of Vienna but these are less than its advantages. It perforce has too strong a hold on the Viennese doctors who are dependent for their living on its courses, and perhaps devote more time than they should to such instruction, good as this instruction may be for the Americans. Also sometimes the American doctors are not of the best type, going as they do to a place where they can get cheap living (about one-half as expensive as in America) and cheap instruction, and can in a short while procure a certificate to take back home with them to launch them in some specialty for which they are not soundly prepared. Also with lack of resources and of sufficient organization the headquarters of the Association are very inadequate and unsatisfactory, located as they are in a cafe. But in answer to these objections I believe it may be stated that the Viennese medical instructors in general do not abuse their connections with the American Medical Association of Vienna and that their courses are mostly excellent. Secondly, the American doctors in Vienna now appear to be in the main earnest and hardworking with a far better morale than I recall in a brief visit to the American Medical Association of Vienna in 1913 when I saw much idleness. And thirdly, the administrative officers have lately been attempting and, in part at least, succeeding in raising the level and standards of the Association, with hopeful plans of better headquarters. Dr. Ralph Reynolds of San Francisco is the president during the present year (1928-1929) and has devoted much time and energy to the improvement of the association.

Unless an American doctor has very special plans of his own and private arrangements for work he can more quickly obtain what he wants by joining the American Medical Association of Vienna than by not doing so. It had been my plan to avoid it until I arrived on the ground when I discovered that the pathology I wanted could quickly be gotten through the association without my being a special burden on the Viennese themselves. Some weeks (three exercises of two hours each a week) were delightfully spent at demonstrations of gross pathology by Professor Erdheim at the Jubiläums Spital

and by Dr. Chiari at the Pathological Institute of the Allgemeines Krankenhaus (Prof. Maresch, Director). Not only was the instruction valuable and stimulating but for me particularly useful since Professor Erdheim's course in itself was an excellent one in medical German by one of the ablest teachers I have ever met, and since a considerable percentage of the pathology in both courses was of the cardiovascular system. Furthermore Dr. Chiari and his associate Dr. Smetana, who spent two years in Baltimore a few years ago, very kindly gave me the opportunity to see the remarkably rich collection of hearts in the Pathological Institute Museum where there is being rearranged this winter one of the finest (perhaps the finest) pathological collections in the world, containing between 5,000 and 10,000 specimens of all kinds, some of them dating back 100 years and more. Visits to the Medicolegal Institute Museum (with its ruptured hearts and aneurysms and its dilated hearts from children dying suddenly with rhachitis), to the Anatomical Institute Museum, and to the Museum of Wax Models in the old Military Medical School were all of considerable interest. It was through the kindness of Dr. Frauendorfer that I saw the Medicolegal collection.

Along with pathology, the study of roentgenology particularly attracted me in Vienna and for a number of weeks I was given valuable instruction in orthodiagraphy and fluoroscopy by Dr. Hugo Rösler (and his associate Dr. Kohn) at the Herzstation. The cardiac roentgenology in Vienna, as I also learned from Dr. Zdansky at the Wenckebach Klinik, is very much developed and well worth observation. Less attention is paid to measurements than to fluoroscopic observation at all angles. The poor financial situation that prevented the taking of plates and films has developed in Europe and perhaps particularly in Vienna, a fine appreciation of the wealth of information that can be obtained by fluoroscopy alone. Orthodiagraphy is in Vienna, as elsewhere on the Continent, the method of choice for recording the heart shadow—a certain amount of practice is of course essential but it seems to be a valuable and not a difficult procedure.

Internal medicine is well represented in Vienna in the various clinics, the First and best known being that of Professor Wenckebach, who is to retire during the coming year; his successor has not yet been chosen. Professor Wenckebach's international reputation and influence have meant much for Vienna especially in the reconstruction days immediately after the war when his friend Sir William Osler helped him to save the medical situation. In spite of his retirement Professor Wenckebach will continue an active interest in medicine, particularly in his chosen field of cardiovascular disease. Associated with Professor Wenckebach in this First clinic are Drs. Porges, Elias, Saxl, who discovered the diuretic property of salyrgan, Hitzenberger, Zdansky, Hofbauer, Freund, Kutschera and Scherf, the last named a keen young man in charge of the "Heart Station" of this clinic as successor to Professor Winterberg, who died prematurely and unexpectedly last month (January, 1929) following an operation. Winterberg had been associated with Rothberger in 1909 when they announced their finding of auricular fibrillation in man a few months before Lewis made the same announcement independently; also Winterberg had been associated with Professor Wenckebach in the publication of their recent work on Unregelmassige Herztätigkeit.

In one of the research laboratories of this First clinic (Wenckebach) there is also now in progress interesting pioneer work in intragastric photography.

Other university medical clinics where undergraduate and graduate instruction is given in Vienna are the Second under Professor Ortner (with Dr. Luger, formerly of the Peter Bent Brigham Hospital, Boston, acting as chief on account of Professor Ortner's illness), the Third under Professor Chvostek and the Fourth under Professor Kovacs. There are also the medical clinics of Professors Pal, Schlesinger, Bauer, Falta, Jagic and Neumann and the Herzstation where Drs. Zak, Dressler, Fischer and Rösler work and teach in heart disease. Dr. Kaufmann who died a year ago was the founder of this heart hospital during the war; it is located in Professor Billroth's old home.

Before closing I must not omit telling of an excellent course of seven lectures of one and one-half hours each given to a few of us in Vienna in German on the History of Medicine by Professor Neuburger, one of the world's authorities on the subject. This was very stimulating, and an added attraction was the demonstration of Laennec's original wooden monaural stethoscope passed on to Vienna from Dr. C. J. B. Williams of London, who had inherited it from Laennec. We listened to each other's hearts with it with much respect and some success. In Vienna we found that the monaural wooden stethoscope is still very popular and in general use, as it had been with Mackenzie. I should add that while we were en route from London to Paris during the course of a week spent in Brittany we were interested to see in the main square of Quimper (Laennec's birthplace in Southern Brittany) between the Townhall and the Cathedral a statue of Laennec seated and holding his stethoscope.

It was to Vienna that Van Swieten and de Haen, leading pupils of Boerhaave, the organizer of the first real medical clinic two hundred years ago in Leyden, came to establish a similar clinic which grew up to a dominating leadership in medicine, with such leaders as Rokitansky and Skoda a hundred years later. Also it was in Vienna that Auenbrugger lived when he proposed percussion as an aid to diagnosis 158 years ago; Professor Neuburger has published a facsimile of Auenbrugger's Latin work on percussion along with early translations into French, English and German.

I hope that these remarks, sketchy though they are, may be of some interest and help to your readers.

Sincerely yours,

PAUL D. WHITE.

AN IMPORTANT FUNCTION OF THE BOSTON MEDICAL LIBRARY

To the Editor of the NEW ENGLAND JOURNAL OF MEDICINE.

Dear Sir:

Reading with great interest the trenchant commentary by Dr. F. E. Rowe in your current issue, I take to myself a certain sense of guilt in that an implied reproach can lie against a campaign which solicits contributions toward the support of a large Medical Library, and makes no promise for the establishment of Branch activities. Apparent justification for this implication must rest in our having failed to make it clear that one of the chief objects which we have in mind is to increase materially the usefulness of the Library to those living at a dis-

tance, through amplified service and Branch establishments.

If it were true that the value of our institution is limited to those living in its immediate vicinity, a verdict of failure might well be adjudged for its existence, and correspondingly the corollary may be stated that only by expansion and extension can it hope to hold and gain support.

To such expansion its policy is pledged.

Truly yours,

JOHN W. BARTOL.

PSEUDO-QUACKS AND REAL FAKERS IN MASSACHUSETTS

Editor of the NEW ENGLAND JOURNAL OF MEDICINE:

In recent months, both the medical and lay press of Massachusetts have on various occasions applied the epithets of "quack" and "faker" to licensed practitioners of the healing art. From the manner in which such epithets are used, one wonders whether those uttering them really appreciate their significance; and how many reading them really understand their meaning?

Indeed, when such characterizations are loosely made by physicians and published so that the lay press thereby obtains opportunity for reprinting them in distorted or inaccurate fashion, may not more harm than good be caused thereby to the entire medical profession?

In order to prevent ambiguity, attention is directed to the definition of these terms in Webster's Unabridged Dictionary.

"Quack—a boasting pretender to medical skill.
—an ignorant practitioner, especially in medicine.
Faker—a pretender—a fraud."

Pursuant to the foregoing definitions, it is contended that licensed practitioners of the art of healing in Massachusetts are not "quacks", nor should such appellation be cast against any of them because so doing invariably re-ëchoes as a boomerang against prominent men selected for the purpose of preventing the licensing of such gentry. The State Board of Registration in Medicine is created, authorized and empowered to see to it that "quacks", viz., pretenders to medical skill, or ignorant practitioners, are not given licenses to practice in Massachusetts. Under the law, applicants are to be subjected to a "comprehensive" examination which is in no manner limited as to scope or thoroughness. When this board has been satisfied as to the competency of a physician, as evidenced by issuing him a license to practice medicine, that fact throws the entire moral and legal responsibility concerning the practicing of licensed "quacks" squarely upon the shoulders of the board. For this reason alone, the unrestrained epithet of "quack" directed towards a legally licensed practitioner is unwise, unjust and unfair to both its recipient and those who gave him the legal right to practice, and should therefore be discouraged.

Conversely, once a practitioner has been licensed, he has the law entirely on his side to the effect that he is not a "quack", and his license is conclusive evidence to that effect which, if reflected upon, means discrediting the entire Board of Registration in Medicine and creates an illogical and untenable position. Moreover, calling a licensed practitioner a "quack" involves professional disparagement which entitles the aggrieved party to damages without requiring specific proof that actual damage has been caused thereby. It is unwise and dangerous practice, and ought not be indulged in by physicians.

Concerning the term "faker", at least in Massachusetts, an unusually peculiar and interesting situation exists in so far as that term may justly be applied to licensed practitioners. If a faker is a pretender and a fraud, certainly we dare not call a licensed practitioner such simply because he is practicing, for in so doing he is lawfully, not fraudulently practicing, and the same dangers are associated with that appellation as apply to the unwarranted use of the word "quack".

On the other hand, there are individuals licensed to practice in Massachusetts who are fakers and pretenders and frauds who are deceiving the public, not because they lawfully practice, but because they pretend to be what they are not—and this in direct violation of a specific Massachusetts statute.

According to the 1927 edition of the American Medical Directory, there are licensed to practice in Massachusetts not less than 136 persons, of whom almost one-half are recorded as never having graduated from any medical school, and the rest as having been graduated from schools not recognized as medical schools by the A. M. A. previous to 1927.

Now, licensing a person to practice in Massachusetts neither directly nor by implication carries with it the right per se to use the title of doctor or M.D. by anyone not lawfully possessing such a degree. Although I have no specific figures at my command, I know that some of the licensed non-graduates are calling themselves doctors, while others append the initials of M.D. to their names without in fact possessing a doctor's degree of any kind. There are also some, who while having doctor's degrees in osteopathy and chiropractic, use their degrees in such a way to imply they are regular M.D.s. These constitute the real fakers among licensed practitioners.

For the foregoing reasons, and to the extent that some licensed practitioners are defrauding the public by pretending that they are doctors when they are not, the Massachusetts statute providing criminal punishment for anyone claiming to hold a degree from an institution of higher learning, when in fact they have not lawfully obtained such, should be invoked. It is not alone a matter of protecting the public from fraudulent representations that this should be done, but in further view of the fact that there are doctors of philosophy, literature, theology, science, law, osteopathy, pharmacy, chiropractic, naturopathy and what not, the term doctor, when applied to practitioners of the healing art, means less and less either as an educational qualification or as a means of designating persons who treat the sick. If the degree of M.D. is to retain its meaning as the highest standard of competency of those practicing the art of healing, then it must be safeguarded from prostitution by fraudulent pretenders.

For the purpose of maintaining that desired standard, no license to practice the art of healing should be issued, or retained in force, excepting under the express condition that holders of the degree of M.D. shall designate themselves as doctors of medicine, and that holders of other doctor's degrees shall likewise distinctly designate what kind of doctor's degrees they hold—and such distinctions shall be made

in a manner so that the public may not be misled into the belief that they are consulting a licensed M.D., when in fact they may be consulting a licensed chiropractor. Those licensed to practice who hold no degrees of doctor, should designate themselves by either appending the title of Mr., or be permitted to call themselves physicians. Such a procedure would effectively weed out the "fakers" from among the licensed practitioners in Massachusetts.

CARL SCHEFFEL, M.D., LL.B.

ADDITIONS TO ARTICLES ACCEPTED BY THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION

Managing Editor,
THE NEW ENGLAND JOURNAL OF MEDICINE.

In addition to the articles enumerated in our letter of February 23, the following have been accepted:

Haley M-O Co.
Magnesia-Mineral Oil (25) Haley
H. K. Mulford Co.
Perfringens Antitoxin-Mulford
National Drug Co.
Diphtheria Toxin-Antitoxin Mixture.
Parke, Davis & Co.
Tetanus-Perfringens Antitoxin, Refined and Concentrated.
G. D. Searle & Co.
Solution Bismuth Sodium Tartrate-Searle, 1.5 per cent. Sulpharsphenamine—Searle
Sulpharsphenamine-Searle, 0.4 Gm. Ampules
Sulpharsphenamine-Searle, 0.5 Gm. Ampules
Sulpharsphenamine-Searle, 0.6 Gm. Ampules

Yours truly,
W. A. PUCKNER, *Secretary*,
Council on Pharmacy and Chemistry.

NEWS ITEMS

RESIGNATION OF DR. HAROLD RAGOLSKY—Dr. Harold Ragolsky has tendered his resignation as Assistant Superintendent at the Lakeville State Sanatorium, Middleboro, Mass. His address after May 1, 1929 will be the Boston City Hospital, Bone and Joint Service.

APPOINTMENT OF DR. C. R. ABBOTT—Dr. Charles R. Abbott of Clinton, Mass., has been appointed and the appointment has been confirmed by the Council, as Associate Medical Examiner for the Fourth Worcester District.

DR. CUSHING'S SIXTIETH BIRTHDAY—About 100 pupils and friends of Dr. Harvey Cushing gathered on Saturday, April 6th, in the Laboratory of Surgical Research, Harvard Medical School, to offer him felicitations on his sixtieth birthday, April 8, 1929. A volume of papers written by his former pupils and published by the *Annals of Surgery* was presented to him by Dr. Elliott Cutler of Cleveland, Ohio. Dr. Cushing spoke about some of the pleasant associations he had had with his pupils in the last thirty years and was deeply moved by this evidence of affection. A note of humor was added to the occasion by the reading of a poem entitled, "The Tie that Blinds" and the presentation of an unusually gorgeous necktie to Dr. Cushing by Dr. Kenneth G. McKenzie of Toronto. Tea was served by Mrs. Cushing and members of the staff of the Peter Bent Brigham Hospital and the Medical School. Among those present, in addition to members of Dr. Cushing's family, were Drs. David Cheever and John Homans of the Peter Bent Brigham Hospital, Dr. Emil Coetsch of Brooklyn, N. Y., and Dr. Samuel C. Harvey of New Haven, Conn.

NOTICES

COURSES IN BRONCHOSCOPY AND ESOPHAGOSCOPY

PARIS, FRANCE, JUNE, 1929

Course I, by Dr. Chevalier Jackson of Philadelphia, at l'Hôpital St-Louis, Service of Dr. Fernand Lemaitre, 10 A. M., June 3rd to 7th inclusive.

This course will consist of five sessions of two hours each. They will be given in English, and will be followed by translations in French by Dr. E. Aucoin and Dr. F. Eeman. The course covers the technique of direct laryngoscopy, bronchoscopy, esophagoscopy and gastroscopy; the mechanical problems of foreign body extraction; the endoscopic diagnosis and treatment of diseases of the larynx, bronchi, lungs, stomach and esophagus. Demonstrations will be by chalk talk, open manikin board, lantern slides and cinematograph.

The session on Tuesday, June 4th, will cover the symptomatology and diagnosis of foreign bodies in the air and food passages from the viewpoint of the general practitioner, the internist and the pediatrician.

The session on Thursday, June 6th, will deal with bronchoscopy as an aid to the internist in the diagnosis and treatment of suppurative diseases of the lungs.

The five sessions of the course are open to physicians and students. No fee is charged and registration is unnecessary.

Course II. The course given by Prof. Chevalier Jackson will be accompanied and followed by a practical course given in English and French by Dr. Edmond Aucoin, Dr. Ellen J. Patterson, Dr. Fernand Eeman, and Dr. Chevalier L. Jackson, assistants of Prof. Chevalier Jackson.

For further information and for registration write to the Secretary, Melle Jeanne Gobréau, 120 Avenue Victor-Hugo, Paris (16e) France.

BULLETIN—BRONCHOSCOPY

The Graduate School of Medicine of the University of Pennsylvania begs to announce to the medical profession that with the opening of the newly built and equipped Chevalier Jackson Bronchoscopic Clinic of the Graduate Hospital of the University of Pennsylvania, it is prepared to continue the teaching of peroral endoscopy. This newest of Bronchoscopic Clinics, which has been designed by Dr. Jackson is a model headquarters for such work, as conducted by Dr. Jackson and his associated staff in the Bronchoscopic Clinics of the two large teaching hospitals of the University of Pennsylvania (University and Graduate Hospitals). The non-clinical portion of the teaching is done in the anatomic and research surgical laboratories of the University.

Such teaching and related research constitutes

a portion of the long courses, one to three years' duration, provided by the Graduate School of Medicine of the University of Pennsylvania, for physicians who pursue studies in the whole field of otolaryngology.

The Graduate School of Medicine, however, has provided a series of two weeks' intensive studies designed for otolaryngologists and surgeons who desire to profit by contact with Dr. Jackson and his Associates in courses for specialists.

TWO WEEKS' COURSES IN LARYNGOSCOPY, BRONCHOSCOPY AND ESOPHAGOSCOPY FOR SPECIALISTS

Future dates for such courses, as now fixed, are: January 28 to February 9, February 25 to March 9, April 8 to April 20, August 5 to August 17, November 4 to November 16, and December 9 to December 21, 1929; January 27 to February 8, February 24 to March 8, and April 7 to April 19, 1930.

The courses are limited to twelve registrants (no one should come to Philadelphia for these courses without having first secured registration by correspondence). They are intended for qualified surgeons or otolaryngologists who desire an intimate view of this very important subject.

Those qualified and desiring registration should address communication: "Dean, Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pa."

GEORGE H. MEEKER, DEAN.

REMOVAL

Dr. D. Harold Walker has removed his office to 5 Bay State Road.

REPORTS AND NOTICES OF MEETINGS

THE CUTTER LECTURE

The Cutter Lecture on Preventive Medicine was delivered at the Harvard Medical School on Wednesday, March 27, 1929 at 5:00 P. M. by Edwin O. Jordan, Professor of Hygiene and Bacteriology at the University of Chicago. His subject was "The Epidemiology of Paratyphoid Infections."

Prof. Jordan first pointed out that there is no recognized clinical differentiation of paratyphoid and typhoid fevers, even agglutination reactions not being conclusive. Only the isolation of the bacteria is accepted as positive proof.

Paratyphoid infection was a common war disease, occurring twice as often as typhoid fever. The attempted explanation was either that typhoid had been replaced by paratyphoid, or that typhoid inoculation had uncovered the person's resistance to paratyphoid infection. Some believe that many of the summer diarrheas previous to milk pasteurization were caused by the paratyphoid bacteria. Due to the mildness of the disease many cases were not reported. Various investigations have shown that paratyphoid carriers are quite prevalent, and that many of the typhoid cases are paratyphoid. Considering these facts, one concludes that paratyphoid infections are more prevalent than is apparent.

Of the members of the paratyphoid group Prof. Jordan considered five which were closely associated with human illness.

Salmonella Paratyphosus (?) is the first one. Its infections are characterized by an abrupt onset and marked remissions, being clinically indistinguishible from B Typhosus infection. Some Germans believe that S. Paratyphosus is primarily a tropical disease, but the fact of its common occurrence in temperate climate seems to disprove this. Its continued occurrence seems to depend on small epidemics and the number of bacilli prevalent.

S. Schottmülleri (Paratyphosus B.) is spread in a similar manner to S. Paratyphosus. Carriers seem to play a large part.

S. Aertrycke is a closely related organism to S. Schottmülleri, but while infections with S. Schottmülleri are characterized by a mild continued fever, those with S. Aertrycke give an explosive intestinal upset.

S. Enteritidis, commonly known as Gärtner's bacillus, causes a violent transient gastro-enteritis. This disease is chiefly caused by meat infected antemortem with the organism, but it has also been found to be associated with rats and mice who harbor and transport it.

S. Suipestifer is found in no other animal except swine. It is the cause of great many epizootics throughout the world; as far as is known the Paratyphosus C. which affects man, is closely related to it.

These five different members of the paratyphoid group are often mistakingly identified. This is due to: (1) mixed cultures being sent out from stock collections; (2) lack of realization that bacilli from swine are not always S. Suipestifer; (3) the fact that S. Schottmülleri, Aertrycke, and Suipestifer all may agglutinate in the same serum and in like concentrations; (4) the fact that there are agglutinative variants because we are unable to control smooth and rough variations.

Prof. Jordan then discussed several general facts. S. Paratyphosus is usually a human organism, rarely an animal one. The objection that we cannot combat its infections because of its prevalence is nullified by the fact that it is not found to be more ubiquitous than other pathogenic bacteria. Second, a brief incubation period in food poisoning suggests a preformed toxin. The same picture cannot be produced in experimental animals or in volunteer humans with heat-killed cultures, thus suggesting a thermolabile toxin. Third, infection with all these organisms is by the usual routes of water, milk, and direct infection. In the case of paratyphoid infections the environment, the season, the susceptibility of the host seem to have great influence on the prevalence.

The specific protection against these infections is best secured by adequate cooking, refrigeration, and the use only of pure meats.

THE LAWRENCE MEDICAL CLUB

March 21, 1929.

The Monthly Meeting of the Club was held Monday evening March 25, with Frank A. Conlon, M.D., of Lawrence at the Nurses' Home, Lawrence General Hospital. Chairman for the Evening, Ernest P. Fuller, M.D., of Lawrence. Subject: Electro-Therapy by Mr. H. C. Morris of Boston.

Two reels of motion pictures of operations upon humans using an electrically charged knife were shown.

THE HARVARD MEDICAL SOCIETY

The Harvard Medical Society held a stated meeting in the amphitheatre of the Peter Bent Brigham Hospital on the evening of March 26, 1929. Dr. H. A. Christian presided. The program consisted of the presentation of cases and Dr. E. P. Joslin introduced the speakers.

The first case was presented by Dr. Deery of the neurological service. The patient was a 45-year old factory foreman, who on routine examination in 1921 was found to have a trace of sugar in his urine. After successful treatment by his local doctor he went to the Massachusetts General Hospital where he was pronounced a mild diabetic. He had been well up to a year ago when he noticed general weakness and occasional lapses of memory. He went to the Deaconess Hospital where in addition to being a mild diabetic, he was noticed to have a blurring of the temporal nervehead. On admission to the P. B. B. H. his physical examination was negative except for a choked disc of 1-2 D which increased to 3 D in four days. A right-sided temporal decompression was done, but this failed to relieve the choking. Ventriculography showed displacement of the lateral ventricles to the right. On operation, which lasted eight and a half hours, a large parasagittal meningioma was removed with difficulty. For two days the patient did well and then became comatose. The diabetes was found not to be a factor because the urine, blood sugar and CO_2 were normal, he is gradually recovering.

The second case was presented by Dr. Rennie of the medical service. The patient was a 55-year old Syrian-born woman who entered the hospital complaining of severe itching of her entire body of seven months' duration. Her family history is negative for diabetes. Eleven years ago she began to have generalized pain throughout her body in the form of pin and needle pricks. She saw several doctors who all diagnosed her as diabetic and gave her diets. She followed these till her symptoms improved and then disregarded them. She has lost 67 lbs. in the last eleven years and her strength has progressively decreased. A year ago she noticed polyuria, nocturia, polydipsia, and boulimia. Two weeks ago she coughed up several teaspoonfuls of bright red blood; this recurred five days later, though at no other time did she have cough or sputum. Physical examination on entry showed an emaciated woman with markedly excoriated skin covered with numerous small infections. Heart and lungs were negative. Her urine showed a trace of sugar, and her blood sugar was 285 mgms. On a diet of 80 gm. carbohydrate, 60 gm. protein, and 120 gm. fat she has been kept sugar free. X-ray of her lungs showed probable tuberculosis in the right upper lobe.

The first paper on "The Pathology of Diabetes" was presented by Dr. Shields Warren. The main pathology of diabetes may be summarized as follows: (1) the pancreas is small, considerably fibrosed, and shows granular atrophy and hyalinized islands; (2) the kidney shows deposits of glycogen in Henle's loops, which may be the origin of the glycosuria; (3) the liver cells show infiltration of their nuclei by glycogen. The use of insulin has disturbed this picture, increasing the normal glycogen deposits and causing the abnormal to disappear. The important problem is not the diabetes but the complications, chief among them being arteriosclerosis and sepsis. The arteries show marked intimal changes of the atheromatous type. The changes in the pancreas are not acute, but are the result of the long struggle between regenerative and degenerative influences exemplified by island proliferation and encroaching hyalinization, respectively.

Miss Hazel Hunt presented the second paper on "Cholesterol and Diabetes." Miss Hunt pointed out that cholesterol determinations give an indication of the blood fat of which 50% are fatty acids, 30% cholesterol, and 20% phosphatides. Any variations in cholesterol indicate equivalent variations in the fatty acids. The more severe the diabetes in patients in the pre-insulin era of therapy, the greater the disturbance of blood lipids and the higher the cholesterol. Nothing under 230 mg/100 cc. is considered high. Low cholesterol levels are reached in 60% of insulin treated patients, the insulin does not interfere with the normal blood ratio. Patients with high cholesterol values may be divided into three groups on the basis of their cholesterol levels: those in coma, the post-adolescent cases, and those unclassified. The significance of this is that control of blood cholesterol may have some influence in preventing the premature development of arterio-sclerosis.

In the third paper Dr. Priscilla White presented "Diabetic Coma—a report of 105 successive cases." Fatalities from coma occur in three types of cases: (1) from uncomplicated coma; (2) coma complicated by infections; (3) death from infection following recovery from coma. In the treatment of coma insulin should be given early and in repeated doses, 20-50 units every half hour in adults. An important secondary factor in cases with upper respiratory infections, that of laryngeal obstruction, must be watched for. The lack of gastric tone must also be combated. Renal trauma is indicated by casts and an increase in the non-protein nitrogen of the blood. The prognosis is good if no overwhelming infections occur and insulin is given before coma goes too far.

Dr. H. F. Root presented a paper on "Resistance to Insulin (Refractory Diabetes)." There are no cases in the literature, said Dr. Root, in which insulin has failed to reduce the blood sugar, though there are many cases where very large doses were necessary. One looks to other processes in the body which may interfere with the functioning of insulin. The resistance to insulin may depend on: (1) complicating diseases, such as cancer, hemochromatosis and acute pancreatitis which lead to destruction of the pancreas; (2) endocrine disorders, notably hyperthyroidism and hyperpituitarism; (3) disturbance of liver function, as in cardiac decompensation, cirrhosis of the liver and syphilis; (4) abnormalities in function of the skin and muscle. The sugar still appearing in the urine may also be due to renal glycosuria.

Dr. Root, taking the place of Dr. W. Stanley Curtis who was unavoidably absent, also spoke on "Heredity and Diabetes." Of 13 cases appearing in twins, ten were in homologous and two in heterologous twins. If diabetes were a true recessive factor, a history of diabetes in the family would be obtained in about 25% of the cases, as is actually found at present. However, as patients are followed over a period of years, so many new cases appear in their families, that the hereditary factor becomes more and more important.

The final paper was presented by Dr. E. P. Joslin on "Gains in Tolerance for Carbohydrate during

Treatment with Insulin". The impression gained in looking over records is that the longer diabetics live, the milder they become. Statistics over a period of years prove that tolerance is not always lost, but it often increases. The carbohydrate in the diet may be increased and the necessary dose of insulin decreased, so that the carbohydrate may rise to 6 grams and even more per unit of insulin.

Dr. Fitz suggested that perhaps the increased tolerance is apparent rather than real in that the hydrogen ion concentration of the medium is brought nearer to the optimum for the action of insulin.

CENSORS' MEETING

The Censors of the Hampden District Medical Society will meet for examination of candidates at Springfield Academy of Medicine, 20 Maple Street, Springfield, on Thursday, May 2, at 4 P. M.

HERVEY L. SMITH, *Secretary.*

249 Union Street, Springfield.

NEW ENGLAND ROENTGEN RAY SOCIETY

The April meeting of the New England Roentgen Ray Society will be held at the Boston Medical Library on Friday, April 26. Dr. H. M. Imboden of New York City will address the society on "Chronic Intermittent Duodenal Stasis".

Dr. Imboden is President-elect of the American Roentgen Ray Society, and one of the leading men in his specialty in the country.

Physicians and medical students are privileged to attend.

M. C. SOSMAN, *Secretary-Treasurer.*

ANNUAL MEETING OF THE MASSACHUSETTS TUBERCULOSIS LEAGUE

The fifteenth Annual Meeting of the Massachusetts Tuberculosis League will be held at the University Club, Stuart Street and Trinity Place, Boston, on Monday, April 22.

At the morning session which will begin at 10:00 o'clock Dr. Frederick T. Lord, Acting President of the League, will preside and will give the President's Annual Address.

Dr. S. A. Petroff, Director, Research and Clinical Laboratory of Trudeau Sanatorium, Saranac Lake, New York, will speak on "Variability of the Tubercle Bacillus". This paper will be illustrated with lantern slides.

Also at the morning session reports of Frank Kiernan, Executive Secretary, and Miss Jean V. Latimer, Educational Secretary of the League, will be presented.

At noon the Annual Meeting of the Corporation will be held at which Directors and Officers of the League will be elected.

At one o'clock the annual luncheon meeting for Directors, Officers and others will be held.

ANNUAL MEETING OF THE BRISTOL SOUTH DISTRICT MEDICAL SOCIETY

April 13, 1929.

The annual meeting of the Bristol South District Medical Society will be held in New Bedford May 2, 1929, at the New Bedford Public Library at 5 P. M.

Dr. Arthur L. Chute of Boston will address the Society. Subject: Urinary Stone. Its Recognition and Treatment. Illustrated.

DR. GEORGE E. BORDEN, *Sec'y.*

132 Franklin Street, Fall River, Mass.

THE HARVARD MEDICAL SOCIETY

The next regular meeting of the Harvard Medical Society will be held in the amphitheater of the Peter Bent Brigham Hospital at 8:15 on the evening of Tuesday, April 23. In addition to the usual presentation of cases, there will be presented through the courtesy of the American Society for the Control of Cancer The Canti Cinematograph Demonstration of Living Tissue Cells Growing in the Vitro and The Effect of Radium Emanation on this Tissue.

WILLIAM P. MURPHY, *Secretary.*

CENSORS' MEETING

The Censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, May 2, 1929 at 4.00 o'clock.

Candidates should make personal application to the Secretary, and present their medical diploma at least one week before the examination.

ARTHUR H. CROSBIE, *Secretary.*

520 Commonwealth Avenue, Boston.

SOCIETY MEETINGS

Combined Meeting of the Boston Medical Library, the Suffolk District Medical Society, the Middlesex South District Medical Society and the Norfolk District Medical Society (at Boston Medical Library at 8:15)

April 24, 1929—Annual meeting. Speaker, Dr. Henry K. Pancoast, Professor of Rontgenology at the University of Pennsylvania. His subject will be "Radiation in Cancer."

The medical profession is cordially invited to attend this meeting.

April 18—The Clinical Conference of the New England Hospital for Women and Children. Detailed notice appears on page 793, issue of April 11.

April 22—Beth Israel Hospital. Detailed notice appears on page 793, issue of April 11.

April 22—The Annual Meeting of the Massachusetts Tuberculosis League. Detailed notice appears elsewhere on this page.

April 23—Harvard Medical Society. Detailed notice appears above.

April 26—New England Roentgen Ray Society. Complete notice appears elsewhere on this page.

May 5-10—First International Congress on Mental Hygiene. Complete notice appears on page 411, issue of February 21.

June 13—International Hospital Congress. Complete notice appears on page 212, issue of January 24.

July 9—American Heart Association. Scientific Meeting from 2 to 6 P. M. in the Auditorium of the Woman's Club, 448 Taylor Street, between 12th and 13th Streets, Portland, Oregon.

October 7-19—New York Academy of Medicine. Detailed notice appears on page 468, issue of February 28.

DISTRICT MEDICAL SOCIETIES

Barnstable District Medical Society

Meeting: May 2, 1929.

Bristol North District Medical Society

April 18, 1929—Spring meeting.

Bristol South District Medical Society

May 2—Annual Meeting. Detailed notice appears elsewhere on this page.

Essex North District Medical Society

May 1, 1929 (Wednesday)—Annual meeting at Lawrence General Hospital, One Garden Street, Lawrence, at 12:30 P. M. (Telephone 5166.)

May 2, 1929 (Thursday)—Censors meet at Hotel Bartlett, 95 Main Street, Haverhill, at 2 P. M. (Tel. 3430.)

Candidates should present their diplomas one week in advance to the Secretary, J. Forrest Burnham, M.D., 567 Haverhill Street, Lawrence.

Essex South District Medical Society

Thursday, May 2, 1929—Censors meet at Salem Hospital, 3:30 P. M. for examination of candidates. Candidates should apply to the Secretary, (Dr. R. E. Stone, Beverly) at least one week prior.

Tuesday, May 14, 1929—Annual Meeting. The Tavern, Gloucester. Speaker: Dr. O. F. Carr, Beverly, Trip around World, Illustrated. Ladies Invited. Dancing.

Franklin District Medical Society

Meeting will be held on the second Tuesday of May.

Hampden District Medical Society

May 2—Censors' meeting. Detailed notice appears on page 851.

Middlesex East District Medical Society

The following schedule has been arranged:

May—Melrose.

Subjects and speakers to be arranged by committees in each city or town.

Middlesex North District Medical Society

The annual meeting will be held the last Wednesday in April.

Norfolk District Medical Society

May—Annual meeting program to be announced.

The Censors of the Society meet May 9, 1929, at the Roxbury Masonic Temple, Warren Street, Roxbury, at 4 P. M., for the examination of candidates.

Plymouth District Medical Society

April meeting at the Commercial Club, Brockton, April 18th, at 6 P. M. Annual Oration by Dr. Ralph C. McLeod, Orator; dinner.

Suffolk District Medical Society

See Combined Meeting in previous column.

May 2—Censors' meeting. Detailed notice appears on page 851.

Worcester District Medical Society

May 8, 1929—Annual meeting. Worcester Country Club.

Worcester North District Medical Society

Annual meeting the fourth Tuesday in April.

BOOK REVIEWS

Roentgenology. The Borderlands of the Normal and Early Pathological in the Skiagram. By ALBAN KÖHLER. Translated from the Fifth German Edition by Arthur Turnbull. Published by William Wood and Company, New York, N. Y. 556 Pages. Price $14.00.

The object of this book was described in the preface of the first German edition which appeared in 1910, "an adviser on those findings which exhibit or appear to exhibit slight and not particularly noticeable divergences from the normal anatomical picture". Every Roentgenologist appreciates the difficulties which so frequently arise in distinguishing normal anatomical variations from pathological processes, and it is in these cases that Köhler will be found a most useful reference.

The first three hundred pages of the book are devoted chiefly to the osseous system, while the remainder of the book deals with the thoracic and abdominal viscera.

The classification of the material is anatomic. As in many German text books on Roentgenology, drawings and sketches have been largely substituted for reproductions of Roentgenolographic negatives. This is highly commendable, and is a lead which might well be followed in many of our own text books.

No one topic is dealt with at any great length, but the reader is referred to some leading article or series of articles on the subject. Thus, as the author states in his preface, the work may be regarded as a lexicon and not suited for systematic study. It is unfortunate for the American student that most of the literature referred to is in German. This very definite drawback has been obviated to some extent by the fact that the translator has introduced in the foot-notes a very considerable number of references from the English and American literature.

The translation at times appears very literal, but it must be appreciated that the task is a difficult one, and in scientific work it is not advisable to deviate too far from the original for the sake of rhetoric.

The book contains a wealth of valuable information, and can be recommended as an addition to the library of all those who are interested in Roentgenology.

Erythema Nodosum. By J. ODERY SYMES, M.D., William Wood and Company, 1928. Price $2.00.

This book of a scant seventy pages discusses "Erythema Nodosum" as seen by a man in general practice over a long period of years. The author regards this condition as an acute specific fever with an incubation period of about fourteen days. On the basis of microscopic examination he submits the thesis that "the lesion is an acute and widespread arteriolitis in the subcutaneous fat lasting a few hours to two days and resolving rapidly without suppuration" and due to "some soluble toxin. Giant cells (fat phagocytes) in large numbers may accumulate in the late stages of a severe lesion. There is nothing to suggest that the local presence of tubercle bacilli accounts for them."

The clinical course is described with emphasis upon the prodromal symptoms. Evidence is presented combating the idea that the disease is a manifestation of acute rheumatism or has an allergic basis and the only relationship to tuberculosis the author can admit is where there is some tuberculotoxemia present, the latter acting like any other toxin. The treatment is non-specific.

Other than the conclusions based on the microscopic examination of tissue in a few cases we are no nearer the etiology than previously. Such studies, however, should be encouraged and are provocative of the thought that a man in general practice can on the basis of experience and interest in one particular phase of a disease perform research which may prove illuminating.

Practical Clinical Laboratory Diagnosis, by CHARLES C. BASS, M.D., and FOSTER M. JOHNS, M.D. Third edition, completely revised. Baltimore, Williams and Wilkins Co., 1929. Pp. 187. $7.50.

This book presents the technique of those laboratory examinations which are useful in ordinary every-day practice, the authors having selected what they consider the best, simplest, and most practical method of making each test. It is obviously a manual for the beginner as it contains 125 text figures, 14 half-tone plates and 6 colored plates, many of which seem superfluous. The cost of these illustrations has made the price of the book what it is.

There is a chapter on the use and care of the microscope with reproductions of different models. The chapter on feces, with its photomicrographs of the ova and larvae of parasitic worms, is excellent. The colored drawings of blood cells, malaria parasites and pathogenic bacteria are works of art. If some of the superfluous illustrations could be omitted and the price reduced, it is certain that this work would have a wider sale.